proportions than hypertensive members of other populations. An alternative explanation for these percentages would be that they are artificially inflated by a downwardly biased denominator, i.e. that the clinical data are inaccurate.

Altering Sample Specifications

We wondered to what degree the estimates we calculated were sensitive to our specific sample specifications and measure of high blood pressure. Consistent with the aims of our larger research program, we had restricted our sample to menstruating women and had used a measure of high blood pressure (MAP) that is commonly used in the obstetric/gynecological literature[26,27], but is not as common in other areas of clinical practice or research.

In Table 4, we report hypertension prevalence estimates (systolic ≥ 140 or diastolic ≥ 90) by age for all Mexican American and Puerto Rican women in HHANES, ages 12–54. Data on medicine usage, history, and a measure that combines clinical and medicine usage data are also reported for these women, as are data for Whites, Blacks and Hispanics in NHANESII. With these changes, we expected rate increases, because MAP is a conservative measure of hypertension that excludes some borderline hypertensives. As expected, the new estimates are higher than those using our previous sample specifications and measure, especially at the older ages where menopausal women have been added. However, the contrasts between clinical hypertension status versus hypertension history remain large. The clinical rates for Puerto Rican and Mexican American women in HHANES remain much lower than the White or Black rates, in each case being no more than one-half the White rate. Again, applying the new specifications to data from NHANESII suggests a different picture from HHANES, suggesting that our initial findings were not simply an artifact of the original specifications.

A Possible Explanation

Great effort was put into quality control when the HHANES data were collected, but our findings raise the question of whether some sort of study effect might explain the low rates of elevated blood pressure readings among examinees. One possibility is the fact that only a few physicians were relied upon to perform the majority of the clinical examinations. This practice has the potential to influence the validity of hypertension data. In general, blood pressure readings have been observed to be subject to variation between examiners.[28,29] This is not to suggest that some physicians' readings are incorrect, but only that there is some expected variation in reporting around "true scores." If there were many physicians evenly distributed across cases, the physician effects would tend to cancel out. However, a reliance on a small number of physicians introduces an extra element of sampling error, one that will not necessarily be accounted for by the calculation of standard errors.[30] As the National Center for Health Statistics has, itself, warned in the past, this practice can seriously jeopardize the validity of a survey.[29]

Table 5 shows the distribution of physicians across the Mexican American and Puerto Rican surveys in our initial sample. Each survey used only four physicians. The distribution of these examiners is uneven, particularly in the Mexican American sample. In the Mexican American survey, Doctors 501 and 502 examined 96 percent of the cases. In the Puerto Rican sample, the examinations are spread across three doctors who, as a group, performed 98.7 percent of the readings.

As shown in the Appendix, we performed sample calculations to estimate the potential statistical inefficiency that might have resulted from the usage of so few examining physicians in HHANES (effectively two for the Mexican American sample and three for the Puerto Rican). The magnitude of the inefficiency produced by using so few physicians depends on both the ratio of the sample size to the number of physicians and on the magnitude of the physician effects. Our estimates suggest that standard errors could have been enlarged three-fold for Puerto Ricans and six-fold for Mexican Americans by the employment of so few physicians.

We conclude from these sample calculations that there appears to be enough variability between examining physicians in the proportion they are likely to diagnose as hypertensive that using only two or three physicians has the potential to reduce substantially the reliability of the survey. However, we present this only as one possible explanation for the low prevalence estimates we found. The data do not permit us to test or rule out other possible systematic biases. We cannot know how likely the physician effects explanation is relative to alternative explanations based on other potential sources of systematic bias.

TABLE 4—Selected Measures of Hypertension by Race and Ethnicity (Clinical HPB Defined as Systolic ≥140 or Diastolic ≥90) All Women in HHANES or NHANESII

	Mexican American				Puerto Rican				White				Black			
Age (years)	CI[a]	CI+M	M[b]	Hx[c]	CI	CI+M	M	Hx	CI	CI+M	M	Hx	CI	CI+M	M	Hx
<15	0.0	0.0	0.0	1.7	0.0	2.5	2.5	3.3	2.9	2.9	0.0	2.0	3.2	3.2	0.0	2.4
15–24	0.3	0.9	0.6	8.1	0.0	0.0	0.0	8.8	2.7	3.1	0.3	6.0	6.3	7.3	1.1	11.5
25–34	3.5	5.2	2.6	17.7	0.5	2.0	1.5	18.3	4.3	5.5	1.5	13.3	12.6	15.1	3.7	18.8
35–44	4.9	7.5	4.2	18.1	5.0	7.2	2.7	26.5	15.2	17.4	5.0	21.0	30.9	37.5	13.9	39.7
45–54	17.6	24.1	12.6	35.1	17.2	24.1	15.4	42.6	28.9	36.1	15.4	29.6	52.6	66.1	40.2	60.2
Totals:																
HHANES	5.2	7.5	4.0	16.9	4.3	6.6	4.0	20.4								
(Standard Errors)	(.5)	(.6)	(.4)	(.8)	(.7)	(.1)	(.7)	(1.3)								
NHANESII	12.1	13.2	2.8	11.2	13.2	13.2	4.3	12.7	10.4	12.6	4.3	14.7	21.0	25.7	11.4	27.1
(Standard Errors)	(2.6)	(2.7)	(1.3)	(2.5)	(4.9)	(4.9)	(3.0)	(4.9)	(.5)	(.5)	(.3)	(.6)	(1.6)	(1.7)	(1.3)	(1.8)

a)CI = Clinical High Blood Pressure
b)M = Antihypertensive Medicine Usage
c)Hx = History of Hypertension

TABLE 5—Distribution of Doctors According to Sample

Doctor	Sample Mexican American	 Puerto Rican
500	3.4	0.0
501	54.9	0.0
502	41.5	40.2
504	0.0	21.2
505	0.0	37.3
510	0.2	1.3
Sample Size	1700	757

SOURCE: HHANES, 1982–84

Conclusions

The several features of the HHANES hypertension data for women we have described suggest that it is wise to be wary of these data and exercise caution when interpreting them. The prevalence of elevated readings at the HHANES examination is low compared to analyses using other data. These examination data appear more discrepant with the history data than is true in NHANESII or that can be accounted for reasonably by recall bias. The validity of the HHANES clinical high blood pressure data may be threatened by the reliance on only a few examining physicians.

Hispanic groups in the US are understudied populations. Given this lack of benchmark data to which we would compare the HHANES data under ideal circumstances, we cannot conclude definitively that the low rates of elevated blood pressure readings estimated for reproductive-age women in HHANES are invalid. However, careful review of the available information reveals inconsistencies that suggest to us that hypertension prevalence estimates based on the HHANES examination data are downwardly biased.

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REFERENCES

1. Subcommittee on Definition and Prevalence of the 1984 Joint National Committee: Hypertension prevalence and the status of awareness, treatment, and control in the United States. Hypertension 1985; 7:457–468.
2. National Center for Health Statistics: Blood pressure levels in persons 18–74 years of age in 1976–80, and trends in blood pressure from 1960 to 1980 in the United States. Vital and Health Statistics, Series 11, No. 234. DHHS Pub. No. PHS 86-1684. Washington, DC: Govt Printing Office, July 1986.
3. Apostolides A, Cutter G, Daugherty S, *et al:* Three-year incidence of hypertension in thirteen US communities. Prev Med 1982: 11:487–499.
4. Stern M, Gaskill S, Allen C, Jr *et al:* Cardiovascular risk factors in Mexican Americans in Laredo, Texas. Am J Epidemiol 1981; 113:546–555.
5. Hanis C, Ferrell R, Schull W: Hypertension and sources of blood pressure variability among Mexican Americans in Starr County, Texas. Int J Epidemiol 1985; 14:231–238.
6. Franco L, Stern M, Rosenthal M, *et al:* Prevalence, detection, and control of hypertension in a biethnic community. Am J Epidemiol 1985; 121:684–696.
7. Kraus J, Borhani N, Franti C: Socioeconomic status, ethnicity, and risk of coronary heart disease. Am J Epidemiol 1980; 111:407–414.
8. Barrios E, Iler E, Mulloy K, Goldstein J, Chalfin D, Munoz E: Hypertension in the Hispanic and black population in New York City. J Nat Med Assoc 1987; 79:749–752.
9. Davis C, Haub C, Willette J: US Hispanics: Changing the face of America. Popul Bull 1983; 38:1–43.
10. Yankauer A: Hispanic/Latino-What's in a name? Am J Public Health 1987; 77:15–17.
11. Hayes-Bautista D, Chapa J: Latino terminology: Conceptual bases for standardized terminology. Am J Public Health 1987; 77:61–68.
12. National Center for Health Statistics: Plan and operation of the Hispanic Health and Nutrition Examination Survey, 1982–1984. DHHS Pub. No PHS 85-1321. Washington, DC: Govt Printing Office, September 1985.
13. National Center for Health Statistics: Plan and operation of the Second National Health and Nutrition Examination Survey, 1976–1980. DHHS Pub. No. PHS 81-1317. Washington, DC: Govt Printing Office, July 1981.
14. Beevers DG, MacGregor GA: Hypertension in Practice. London: Martin Dunitz Publishers, 1987.
15. Burton AC: Physiology and Biophysics of the Circulation: An Introductory Text. Chicago: Yearbook Medical Publishers, 1972.
16. Kalton G: Practical methods for estimating survey sampling errors. Bull Int Stat Inst 1977; 47:495–514.
17. Rust K: Variance estimation for complex estimators in sample surveys. J Official Stat 1985; 1:381–397.
18. Kovar MG: Approaches for the analysis of data. *In:* National Center for Health Statistics: Plan and Operation of the Hispanic Health and Nutrition Examination Survey, 1982–1984. Vital and Health Statistics, Series 1, No. 19. DHHS Pub. No. PHS 85–1321. Washington, DC: Govt Printing Office, September 1985.
19. Landis J, Lepkowski J, Eklund S, Stenhouwer S: A statistical methodology for analyzing data from a complex survey: The first National Health and Nutrition Examination Survey. Vital and Health Statistics, Series 2, No. 92. DHHS Pub. No. PHS 82–1366. Washington, DC: Govt Printing Office, September 1982.
20. Survey Research Center Computer Support Group: OSIRIS IV User's Manual. Ann Arbor: Institute for Social Research, University of Michigan, 1979; 195–202.
21. Kovar MG, Johnson C: Design effects from the Mexican American portion of the Hispanic Health and Nutrition Examination Survey: A strategy for analysts. 1986 Proceedings of Section on Survey Research of the American Statistical Association.
22. Lago J, Massey J, Ezzati T, Johnson C, Fulwood R: Evaluation of design effects for the Mexican American portion of the Hispanic Health and Nutrition Examination Survey, 1982–1983. American Statistical Association presentation, August 1987.
23. Division of Health Examination Statistics, National Center for Health Statistics (NCHS): Analytic Guidelines and Reliability Criteria for Analyzing HANES data. Hyattsville, MD: NCHS, May 1989.
24. Shryock HS, Siegel JS: The Methods and Materials of Demography. Washington, DC: Govt Printing Office, 1971; 289–291.
25. US Department of Health and Human Services: Report of the Secretary's Task Force on Black and Minority Health: Executive Summary. Washington, DC: Govt Printing Office, August 1985.
26. Chesley LC, Sibai BM: Clinical significance of elevated mean arterial pressure in the second trimester. Am J Obstet Guynecol 1988; 159:275–279.
27. Villar L, Sibai BM: Clinical significance of elevated mean arterial blood pressure in the second trimester and threshold increase in systolic or diastolic blood pressure during third trimester. Am J Obstet Gynecol 1989; 160:419–423.
28. Neufeld PD, Johnson DL: Obser[illegible] in blood pressure measurement. CMAJ 1986; 135:633–637.
29. National Center for Health [illegible]server variability in blood pressure measurement. Appen[illegible]Pressure of Adults by Age and Sex: United States-1960–1962. [illegible]tatistics, Series 11, No. 4, Public Health Service Pub[illegible]gton, DC: Govt Printing Office, June 1964.
30. Cochran WG: Sampling Techniques. [illegible]Wiley & Sons, 1977.

APPENDIX

The magnitude of the inefficiency due to the reliance on few examining physicians depends on both the ratio of the sample size to the number of doctors and on the magnitude of the doctor effects. In particular, imagine that *n* doctors each examine *m* respondents. Also, let the true population proportion with hypertension be π, while the probability that the i_{th} doctor identifies a person as hypertensive is π_i. While we might assume that

$$E_i(\pi_i) = \pi,$$

the fact that there are doctor effects implies that, in general, $\pi_i \neq \pi$. A natural measure of the magnitude of these effects is just the variance of the π_i's,

$$\sigma^2_{\pi_i} = E_i(\pi_i - \pi)^2.$$

Were it not for the doctor effects the sampling variance of p, the proportion of respondents with hypertension, on a sample of size $N = mn$ would just be

$$\frac{\pi(1 - \pi)}{N}.$$

Doctor effects add an extra component to this variance. With doctor effects the variance will be

$$\frac{E_i[\pi_i(1 - \pi_i)]}{N} + \frac{\sigma^2_{\pi_i}}{n}.$$

Thus the efficiency of the survey design relative to that of a simple random sample is just:

$$\frac{\dfrac{E_i[\pi_i(1 - \pi_i)]}{N} + \dfrac{\sigma^2_{\pi_i}}{n}}{\dfrac{\pi(1 - \pi)}{N}}$$

Note if either the number of doctors used, n, is larger or doctor variability, $\sigma^2_{\pi_i}$ is small the above expression approximates 1 implying that in these circumstances the inefficiency is small.

To evaluate this expression we need an estimate of $E_i[\pi_i(1 - \pi_i)]$ and $\sigma^2_{\pi_i}$. With effectively only two doctors in the Mexican and three in the Puerto Rican samples there are too few doctors to allow a reasonable estimate of $\sigma^2_{\pi_i}$. What we can do, though, is use the sample of Whites in NHANESII to calculate sample analogies for the above expressions. From these we can estimate the impact that using only two or three doctors would have on the efficiency of the NHANESII survey.

In the NHANESII survey the 2,963 White women were surveyed by a total of 13 doctors. The sample variance,

$$S^2_{\pi_i} = \frac{\sum_i(p_i - \bar{p}_i)^2}{12}$$

is .00230. The sample variance reflects both systemic differences between the doctors and sampling variability. To get an estimate of $\sigma^2_{\pi_i}$ we need to subtract off from $S^2_{\pi_i}$ an estimate of the sampling variability. This we can estimate as

$$\frac{\sum_i\left[\dfrac{p_i(1 - p_i)}{m_i}\right]}{n}$$

= .00046. Thus a calculation gives us an estimated $\sigma^2_{\pi_i}$ of .00230 − .00046 = .00184. To estimate $E_i[\pi(1 - \pi)]$ we simply use

$$\frac{\sum_i p_i(1 - p_i)}{12}$$

= .04580.

Now suppose that we were to redo the NHANESII survey using only two doctors, each of whom tests 800 respondents (similar to the distribution in the Mexican sample). Evaluating the above formula implies that the efficiency of the random sample is 35 times that of the sample using only two doctors. In other words, the standard errors are almost six times what they would have been with random sampling. Redoing the calculation assuming that three doctors each do 250 cases (similar to the distribution in the Puerto Rican sample), we find that a random sample with the number of respondents would be roughly 11 times as efficient or the standard errors are more than three times as large as they would be with a random sample. We conclude from these samples calculations that there appears to be enough variability between physicians in the proportion they are likely to diagnose as hypertensive that relying on only two physicians to examine the Mexican American women and only three for the Puerto Rican women has the potential to reduce substantially the survey's reliability.

Fruit and Vegetables in the American Diet: Data from the NHANES II Survey

Blossom H. Patterson, MA, Gladys Block, PhD, William F. Rosenberger, MPhil,
David Pee, MPhil, and Lisa L. Kahle, BA

Abstract: Twenty-four hour dietary recall data from the Second National Health and Nutrition Examination Survey (1976–80) were used to estimate the numbers of servings of fruit and vegetables consumed by Black and White adults, to examine the types of servings (e.g., potatoes, garden vegetables, fruit, and juice), and to estimate the mean intake of calories, fat, dietary fiber, and vitamins A and C by number of servings. An estimated 45 percent of the population had no servings of fruit or juice and 22 percent had no servings of a vegetable on the recall day. Only 27 percent consumed the three or more servings of vegetables and 29 percent had the two or more servings of fruit recommended by the US Departments of Agriculture and of Health and Human Services; 9 percent had both. Consumption was lower among Blacks than Whites. The choice of vegetables lacked variety. Diets including at least three servings of vegetables and two servings of fruit contained about 17 grams of dietary fiber. Although caloric and fat intake increased with increasing servings of fruit and vegetables, the percent of calories from fat remained relatively constant. Although these data are 10 years old, more recent surveys have shown similar results. The discrepancy between dietary guidelines and the actual diet suggests a need for extensive public education. (*Am J Public Health* 1990; 80:1443–1449)

Introduction

In a recent article,[1] it was found that a large proportion of the adult United States population eats no vegetables (0.17) or fruit (0.41) on any given day. The Surgeon General's Report on Nutrition and Health[2] recommends increasing consumption of vegetables and fruit. Based on a comprehensive literature review, the National Academy of Sciences (NAS) concluded that diet influences the risk of several major chronic diseases and recommended eating five or more daily servings of a combination of vegetables and fruit, especially green and yellow vegetables and citrus fruit.[3] The US Department of Agriculture (USDA) and the Department of Health and Human Services (DHHS) recommend as part of their food guidance system that the daily diet include two to three servings of fruit and three to five servings of vegetables.[4,5] These specific recommendations are in the context of general recommendations for a diet that meets nutrient requirements, does not include excessive amounts of fat (in particular saturated fats), and is consistent with maintaining desirable weight. It is important therefore to determine what relation increased fruit and vegetable consumption might have, not only to intake of obviously related nutrients such as vitamins A and C but also to fat intake and maintenance of appropriate body weight. In the NHANES II data, collected between 1976 and 1980, it is possible to examine these relationships in self-selected diets that were actually consumed during a 24-hour period by a representative sample of US adults.

The article mentioned above[1] described the proportions of the US population that consumed any amount of a given food, however large or small; no attempt was made to determine the number of servings consumed. In the present inquiry we estimate the number of servings consumed, taking portion size into account. We also examine the intake of calories, fat, fiber, and vitamins A and C in the diets of persons consuming various numbers of servings of fruit and vegetables. While it is obvious that intake of vitamins A and C would increase with increasing numbers of servings, the relative contribution of fruit versus vegetables is not well known, nor is the nutrient intake actually achieved in self-selected diets. Similarly, the dietary fiber intake contained in diets including multiple servings of fruit and vegetables may be inadequately appreciated by the general public.

Methods

The NHANES II survey was conducted by the National Center for Health Statistics between 1976 and 1980.[6] A highly stratified multi-stage probability design was used to obtain a representative sample of the civilian noninstitutionalized population, ages six months to 74 years; we report on 10,313 White and 1,335 Black adults ages 19 to 74. We excluded other races because of small numbers, as well as imputed, unreliable, or surrogate data. Results are based on weighted data, permitting inference about the total Black and White noninstitutionalized US population.[7] Group means and standard errors were calculated using software appropriate for complex sample survey data.[8] Standard errors are frequently large for Blacks, due to their small number in the survey. Therefore, results for Blacks should be viewed with caution. Conversely, standard errors for Whites are small due to the very large sample size, so that small differences in intake, while not biologically meaningful, could be statistically significant.

Dietary interviews were conducted by interviewers with a knowledge of food preparation and nutrient composition. A measure of portion size was obtained using three-dimensional food models, as well as dishes and glassware of various sizes. Subjects were asked to report all food and drink consumed in the 24-hour period before the interview day. Almost all recall days were weekdays. Food codes, food weight, and nutrient values were based on data from the USDA, industry, and other sources.[9] The coding of these foods has been described elsewhere.[10] Based on reported portion sizes, the number of grams consumed was calculated and included on the 24-hour recall tape[11] for each food reported by each respondent.

Address reprint requests to Blossom H. Patterson, MA, Clinical and Diagnostic Trials Section, Biometry Branch, Division of Cancer Prevention and Control, National Cancer Institute, Executive Plaza North, Rm 344, Bethesda, MD 20892. Dr. Block is also with that Section at NCI; the other co-authors are with Information Management Services, Inc., Silver Spring, MD. This paper, submitted to the Journal August 3, 1989, was revised and accepted for publication May 10, 1990.

We converted the number of grams reported for each fruit and vegetable into a number of servings as follows. We used as a guide suggested serving sizes given by a food guidance system developed to help implement the USDA/DHHS dietary guidelines.[5] For fruits, a serving is defined as an average piece of whole fruit or six ounces of fruit juice, and for vegetables, one-half cup, cooked or raw. An examination of a sample of actual food records showed that some reported amounts of fruit or vegetables, such as a slice of onion on a hamburger, or lemon juice added to tea, were too small to be considered a serving, while others were so large that they should be considered as more than a single serving. We chose a lower limit of one ounce (30 grams) for any serving of a fruit or vegetable, and two ounces (62 grams) for fruit juice. Small lower limits on a single serving were chosen so as not to eliminate portions consumed by persons with small appetites. This choice may result in some overestimation of the proportions eating one or more servings on the recall day. We chose an upper limit for a single serving so that a large portion (eaten on a single occasion) would be treated as more than a single serving. A single portion of a fruit or vegetable was considered to be at most two servings so as not to overestimate the number of servings of big eaters. Juice consumed on a single eating occasion was considered (at most) three servings.

In order to convert a large portion of whole fruit or vegetables into two servings, we determined a representative or "typical" number of grams in a half cup serving of vegetables, and the weight of an "average" piece of fruit, using weights given for the most popular[1] vegetables and fruits in Agriculture Handbook No. 456.[12] A half cup serving of many vegetables weights about 75 grams, and many whole fruits weigh roughly 120 grams. We defined as two servings any portion of vegetables (except salad) weighing 150 grams or more, and any portion of fruit (excluding fruit juice) weighing 240 grams or more. For fruit juice, individuals consuming between 12 and 18 ounces (372 to 557 grams) were given credit for two servings of fruit, and those consuming 18 or more ounces (558 or more grams) were given credit for three servings. An individual eating a green salad on a single occasion was given credit for a single serving rather than for multiple servings corresponding to each ingredient. Thus individuals could be credited with a maximum of one salad per eating occasion, or a maximum of the four different occasions per day coded in the survey.

Individual foods included in each food category are given elsewhere.[1] Vegetables include potatoes, dried peas and beans (e.g., black-eyed peas, kidney beans), green salad (primarily lettuce and raw tomatoes), as well as all other vegetables not falling into one of these categories. This last group is designated "garden vegetables" and includes, for example, green beans, corn, and broccoli. "Fruit" refers to whole fruit and fruit juice. In this article, fruit juices include citrus and other natural fruit juices, but exclude both fortified and non-fortified fruit "drinks." A list is available from the authors on request.

We report mean nutrient intake by number of servings (0, 1, 2, 3 or more) of fruit cross-classified by number of servings of vegetables. Nutrient intake was calculated by the National Center for Health Statistics, using USDA nutrient data, updated 1980. Because consumption of fruit and vegetables is associated with age, sex, and race,[1] we controlled for these factors in each cell of the cross-classification tables using a method equivalent to direct standardization. This method for finding means and the corresponding variance estimates, adjusted for age, race, and sex, is given in the Appendix. The method uses regression coefficients to obtain estimates of means.

Results

Because the NHANES II data were collected before the recommendations above were issued, they do not reflect their impact; rather the guidelines are used as a standard against which to describe the diet. In the spirit of both the USDA/DHHS and NAS guidelines, we define three or more servings of vegetables and two or more servings of fruit as meeting the vegetable and fruit guidelines, respectively. The proportions meeting these guidelines are shockingly small (Table 1). On the recall day, 27 percent met the vegetable guidelines and 29 percent met the fruit guidelines, while only 9 percent met both. In general, proportions were highest among older Whites but, even in this group, fewer than one-third ate the recommended servings of vegetables, and fewer than half ate the recommended servings of fruit. More men had adequate numbers of servings of vegetables than did women; the opposite was true for fruit. About two-thirds as many Blacks as Whites met these guidelines. Only about 5 percent of Black women and 7 percent of Black men consumed the recommended numbers of both fruit and vegetable servings.

Eleven percent ate neither fruit nor vegetables on the recall day, 45 percent had no servings of fruit, and 22 percent had no servings of a vegetable (Table 2). In contrast to the five or more servings of fruit and vegetables recommended, zero or one serving might be considered clearly inadequate. Thirty-five percent of Black adults and 27 percent of White adults had at most one serving of a food in the fruit/vegetable food group (data available from author on request).

Mean numbers of servings of fruit (1.08, SE = .03) and vegetables (1.77, SE = .02) were far below recommended levels and were lower for Blacks than for Whites (data available from author on request). Mean vegetable intake was

TABLE 1—Proportions (standard error) of Persons Ages 19–74 Who Met or Exceeded the USDA Guidelines, by Race, Sex, and Age Group: Estimates Based on Data from NHANES II, 1976–80.

	Age Group (years)			
	19–29	30–54	55–74	All Ages
Fruit[*] and Vegetables[†] Guidelines (2+ Fruits and 3+ Vegetables)				
White Males	.08 (.01)	.09 (.01)	.12 (.01)	.09 (.01)
Black Males	.05 (.01)	.08 (.02)	.08 (.02)	.07 (.01)
White Females	.05 (.01)	.08 (.01)	.15 (.01)	.09 (<.01)
Black Females	.04 (.02)	.03 (.01)	.08 (.02)	.05 (.01)
All	.06 (.01)	.08 (.01)	.13 (.01)	.09 (<.01)
Vegetable Guidelines (3 or more servings)				
White Males	.32 (.01)	.31 (.01)	.31 (.01)	.31 (.01)
Black Males	.22 (.04)	.20 (.04)	.23 (.05)	.21 (.02)
White Females	.21 (.01)	.25 (.01)	.28 (.01)	.25 (.01)
Black Females	.15 (.03)	.18 (.04)	.20 (.03)	.18 (.02)
All	.25 (.01)	.27 (.01)	.28 (.01)	.27 (.01)
Fruit Guidelines (2 or more servings)				
White Males	.21 (.02)	.25 (.01)	.35 (.01)	.26 (.01)
Black Males	.16 (.03)	.24 (.03)	.22 (.03)	.21 (.02)
White Females	.24 (.02)	.28 (.01)	.47 (.02)	.32 (.01)
Black Females	.24 (.05)	.20 (.03)	.32 (.05)	.24 (.02)
All	.22 (.01)	.26 (<.01)	.40 (.01)	.29 (<.01)

[*] Whole fruit and fruit juice

[†] Potatoes, salad, dried peas and beans, all other vegetables (denoted "garden": see text)

TABLE 2—Proportions (standard error) of Persons Ages 19–74 and Numbers in Sample, by Numbers of Servings of Fruit and/or Vegetables Consumed: Estimates Based on Data from NHANES II, 1976–80

Servings of Fruit[*]	Servings of Vegetables[†]				
	0	1	2	3+	All
0	.11 (<.01) 1,306	.11 (<.01) 1,244	.11 (<.01) 1,233	.11 (<.01) 1,247	.45 (.01) 5,030
1	.05 (<.01) 633	.07 (<.01) 814	.07 (<.01) 804	.07 (<.01) 847	.26 (<.01) 3,098
2	.03 (<.01) 325	.04 (<.01) 450	.04 (<.01) 486	.05 (<.01) 562	.15 (<.01) 1,823
3+	.02 (<.01) 261	.03 (<.01) 403	.04 (<.01) 481	.04 (<.01) 552	.14 (.01) 1,697
All	.22 (.01) 2,525	.26 (.01) 2,911	.26 (.01) 3,004	.27 (.01) 3,208	1.000 11,648

[*] Whole fruit and fruit juice

[†] Potatoes, salad, dried peas and beans, all other vegetables (denoted "garden": see text)

somewhat higher for males than females; the opposite was true for fruit and juice. The mean number of servings of garden vegetables, a group including the green and yellow vegetables emphasized by the NAS guidelines, was only 0.65 (SE = .01).

At least one serving of garden vegetables was eaten by 43 percent of the adult population, and there were no notable differences by race or sex (Table 3). Potatoes and salad were more popular among Whites than Blacks. Proportions eating fried potatoes declined with age, while those consuming non-fried potatoes increased (data available on request to author). Dried peas and beans, good sources of fiber, were eaten by only about 10 percent of the population.

About half the population consumed no servings or only one serving of a vegetable (Figure 1). Among those who reported only one serving of a vegetable, salad was the most popular, reported by 39 percent. Twenty-nine percent consumed a serving of potatoes as their only vegetable, and over half of these servings of potatoes were fried. Among those reporting two servings of a vegetable, some did not achieve variety: 9 percent had two servings of potatoes, 7 percent reported two servings of salad, and 5 percent ate two servings of dried peas and beans.

Only slightly more than half of the population (55 percent) had at least one serving of fruit or fruit juice. Of these, 27 percent had juice only, and 45 percent had whole fruit only (data available on request to author).

Table 4 shows average intake of dietary fiber, vitamin A, and vitamin C among persons consuming various numbers of servings of fruit and vegetables. These nutrient levels represent dietary intake from all food sources, but exclude vitamin supplements. Persons who consumed neither fruit nor vegetables had, on average, only 5.6 grams of fiber in their diet on the recall day, or about one-fourth of the 20–30 grams recommended by the National Cancer Institute.[13] The amount of fiber in these self-selected diets increased with the total number of servings of fruit and vegetables consumed, and this increase was similar for both fruit and vegetables. A total of five servings of a combination of fruit and vegetables was associated with an intake of approximately 17 grams of fiber. The recommended level of fiber intake was achieved only by those who had three or more servings of *both* fruit and vegetables, a group representing only 4 percent of the US adult population.

In the United States, it is estimated that the usual foods available to consumers provide about half of the total vitamin A activity in the diet as provitamin A carotenoids, found in plant products, and the other half as retinol.[14] Among those eating both fruit and vegetables, the US Recommended Daily Allowance (RDA) of 5000 IUs of vitamin A was attained only by those having at least two servings, while five servings of a combination of fruit and vegetables were associated with mean vitamin A intake of about 8000 IUs. If either vegetables or fruit were eaten to the exclusion of the other (true of over half the population), the US RDA was reached only among those having three or more servings.

For vitamin C, fruit and vegetables represent the primary source in the US diet,[10] and Table 4 shows that fruit in particular is associated with higher intake. Among those consuming no fruit, the US RDA of 60mg was attained only by those who had three or more servings of vegetables. This is notable in view of the fact that 45 percent of the US population consumed no servings of fruit on the day of the survey.

Table 5 shows caloric and fat intake by servings of fruit and vegetables. Caloric intake increased as numbers of servings of either food increased. However, the additional calories were not due to the fruit or vegetables alone, as fat intake also increased substantially. This is presumably due in part to fats such as butter and margarine added to potatoes and other vegetables and to oils in salad dressings; people who eat more servings of fruit and vegetables may also eat more food in general. The increase in dietary fat intake associated with increased servings of vegetables was greater than that associated with increased servings of fruit (data available on request to author). Those who ate no fruit and three or more servings of vegetables had an average of 95 grams of fat in their diets, while those who ate three servings of fruit and no vegetables had 78 grams.

TABLE 3—Proportions (standard error) of Persons Consuming One or More Servings of a Vegetable, by Race, Sex, and Type of Vegetable: Estimates Based on Data from NHANES II, 1976–80

Type of Vegetable	Whites		Blacks		All
	Males	Females	Males	Females	
Garden Vegetables[*]	.42 (.01)	.44 (.01)	.41 (.03)	.44 (.02)	.43 (.01)
Potatoes					
Fried	.22 (.01)	.13 (.01)	.15 (.02)	.10 (.01)	.17 (<.01)
Not Fried	.28 (.01)	.26 (.01)	.20 (.02)	.20 (.02)	.26 (.01)
All	.46 (.01)	.37 (.01)	.33 (.03)	.30 (.03)	.40 (.01)
Salad	.39 (.01)	.40 (.01)	.19 (.02)	.24 (.02)	.37 (.01)
Dried Peas and Beans	.11 (.01)	.08 (.01)	.13 (.02)	.10 (.02)	.10 (.01)

[*] All vegetables except potatoes, salad, and dried peas and beans. Examples are green beans, carrots, and corn.

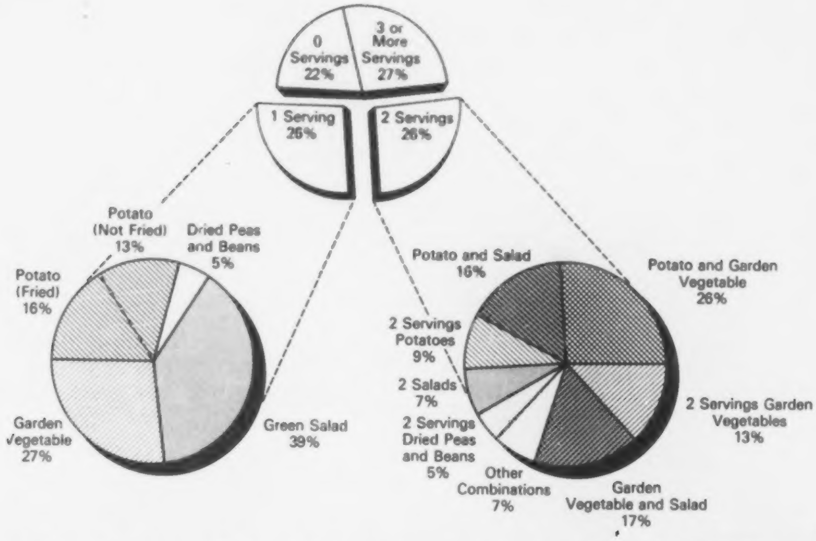

FIGURE 1—The top pie chart shows the percentages of individuals who consumed zero, one, two, or three or more servings of vegetables on the recall day. Percentages are estimates based on data from NHANES II, 1976–80. The lower left pie chart displays the type of vegetable consumed by those who consumed only one serving of a vegetable. The pie chart on the lower right displays the types of vegetables consumed by those who had exactly two servings of a vegetable. The category "garden vegetables" includes all vegetables except potatoes, salad, dried peas and beans. Examples are green beans, carrots, and corn.

When we express fat as a percent of calories, there was a small increase with increasing vegetable intake, approximately two percentage points from the lowest to the highest serving category. In contrast, with increasing fruit consumption, the percent of calories from fat fell approximately five percentage points from the lowest to the highest consumption category. Percent of calories from saturated fat behaved similarly, reaching its highest value among those who consumed no fruit and two or more vegetables, and its lowest value among those who consumed three or more servings of fruit and no vegetables (data available from authors on request).

Although the percent of calories from fat increased slightly with increasing vegetable consumption, this was associated with an increase in the ratio of polyunsaturated fats to saturated fats (P/S ratio), the direction recommended by the American Heart Association[15] (data available from authors on request). This may reflect the use of unsaturated oil in salad dressings.

Despite the increase in caloric intake and dietary fat with increasing numbers of servings of fruit and vegetables, there was no associated increase in obesity (data available from authors on request). A commonly used measure of obesity, the Quetelet Index (weight(kg)/height(m)^2) was relatively constant across all serving categories, ranging from 24.9 (SE = .27) to 25.6 (SE = .18). This may reflect differences in energy expenditure associated with higher caloric levels.

Discussion

"In this land of plenty millions of Americans aren't eating wisely. Not because they haven't enough to eat, but because they eat too many of the wrong things or too little of the right,"[17] according to the USDA. Based on 24-hour recall data, we found that 91 percent of the adult US population did not meet the USDA/DHHS recommendation that two to

TABLE 4—Mean Intake of Various Nutrients (Standard Errors) by Number of Servings of Fruit and Vegetables, Adjusted for Age, Race, and Sex: Estimates based on Data from NHANES II, 1976–80

	Servings of Vegetables[†]			
	0	1	2	3 or more
*Servings of Fruit**	Dietary Fiber (g)			
0	5.6 (.2)	6.62 (.2)	9.7 (.3)	13.8 (.5)
1	7.7 (.2)	8.8 (.3)	10.5 (.3)	14.8 (.4)
2	9.9 (.3)	11.0 (.3)	13.2 (.3)	16.6 (.3)
3 or more	13.8 (.5)	14.7 (.3)	17.2 (.4)	21.0 (.5)
	Vitamin A (IU)			
0	2998 (167)	3333 (189)	4880 (430)	5882 (269)
1	3804 (314)	5364 (649)	5311 (279)	6789 (325)
2	3876 (253)	5372 (451)	5855 (283)	7934 (456)
3 or more	5330 (393)	7041 (403)	8924 (615)	10393 (526)
	Vitamin C (mg)			
0	25.9 (2.1)	38.3 (1.7)	51.4 (1.8)	79.6 (3.4)
1	73.0 (2.0)	95.5 (4.4)	102.7 (2.7)	130.0 (3.7)
2	114.4 (6.9)	123.5 (3.3)	145.0 (4.6)	163.0 (3.9)
3 or more	218.0 (12.0)	211.8 (10.7)	222.8 (7.0)	248.3 (9.7)

USRDA for vitamins A and C is 5000 IU and 60 mg respectively. The National Cancer Institute recommends a daily fiber intake of 20–30g with an upper limit of 35 g.

*Whole fruit and fruit juice

[†] Potatoes, salad, dried peas and beans, all other vegetables (denoted "garden": see text)

TABLE 5—Mean Caloric and Fat Intake (standard errors) by Number of Servings of Fruit and/or Vegetables, Adjusted for Age, Race, and Sex (Estimates based on Data from NHANES II, 1976–80)

	Servings of Vegetables†			
	0	1	2	3 or more
*Servings of Fruit**	Calories			
0	1633 (30)	1817 (26)	1950 (33)	2230 (47)
1	1741 (34)	1890 (36)	1941 (32)	2264 (70)
2	1924 (51)	1937 (48)	2029 (43)	2249 (49)
3 or more	2112 (84)	2137 (59)	2173 (57)	2369 (50)
	Fat *(g)*			
0	64.4 (1.3)	75.6 (1.8)	84.4 (1.8)	95.4 (2.1)
1	69.1 (1.9)	79.7 (1.9)	81.2 (1.6)	98.3 (3.5)
2	75.8 (2.9)	77.2 (2.5)	81.3 (2.3)	93.5 (3.3)
3 or more	77.6 (3.7)	83.2 (3.1)	83.3 (3.0)	92.0 (2.4)
	Percent Calories from Fat‡			
0	37.6 (.3)	40.1 (.4)	40.6 (.3)	40.3 (.3)
1	36.1 (.3)	38.9 (.4)	39.6 (.3)	39.8 (.4)
2	36.1 (.5)	36.5 (.5)	36.9 (.5)	37.8 (.3)
3 or more	32.7 (.7)	35.0 (.6)	34.4 (.5)	34.8 (.4)

*Whole fruit and fruit juice

† Potatoes, salad, dried peas and beans, all other vegetables (denoted "garden": see text)

‡ Calculated as percent of nonalcohol calories.[16]

three servings of fruit and three to five servings of vegetables be eaten daily.

Our method of defining a serving in terms of the number of grams eaten at any given meal, as opposed to the total number of grams consumed during the entire 24-hour period, may have resulted in some bias in the estimation of number of servings. Small portions (weighing less than an ounce) might sum to a serving over the course of a day, resulting in an underestimation. This bias is probably minimal because of the small lower limits (one ounce of fruit or vegetable) used in the estimation. Conversely, these small limits might result in an overestimation of the number of servings; our lower limit is equivalent to less than half of a serving as defined by the USDA. The second bias is likely larger than the first and, if so, the results presented in this article are unduly optimistic.

While a single day's food record cannot be used to characterize an individual's usual diet, 24-hour recall data can provide estimates of group means.[7] Our finding that 11 percent had no servings of either fruit or vegetables on the recall day is in close agreement with data from the NHANES II food frequency questionnaire in which 12 percent reported that they eat fruit and vegetables less frequently than daily, and with 24-hour recall data from the Nationwide Food Consumption Survey (NFCS), conducted by the USDA in 1977–78, in which 10 percent of women and 8 percent of men ages 19–50 reported eating no fruit or vegetables on the recall day.[18]

A comparison of 24-hour dietary recall data among adults ages 19–50 from the NFCS and from the 1985 and 1986 Continuing Survey of Food Intakes (CSFII),[19–21] also conducted by the USDA, suggests that there has been little change in the percentages using these foods. The proportion of men reporting eating any vegetables on the recall day decreased from 89 to 85 percent between 1977 and 1985, while the proportion reporting fruit consumption was virtually unchanged: 44 percent in 1977 and 43 percent in 1985. For women, changes were also minimal: in 1977, on the recall day, 84 percent reported eating a vegetable, compared to 83 percent in 1985; 50 percent reported eating fruit in 1977 and 47 percent in 1985. CSFII data for women in 1986 were similar. A study of food consumption trends based on NFCS and CSFII data reported that proportions of women using green and yellow vegetables and "lower-fiber" vegetables such as lettuce and tomatoes increased slightly during this period, while proportions using "higher-fiber" vegetables such as corn, green peas, and cooked cabbage decreased.[22] While data from the USDA surveys are directly comparable, these surveys differ somewhat from the NHANES II survey in sampling and in data collection methods. Further, the USDA vegetable group is slightly more comprehensive than ours, and their fruit group excludes some citrus fruit juices, such as lemonade, that were included in our study. Recent promotion of dietary fiber may have produced some increase in the consumption of fruit and vegetables, but is unlikely to have resulted in changes of the magnitude needed to meet the guidelines.

Results of a small survey (298 adults selected by random digit dialing) conducted in two counties in California in 1987[23] were similar to our findings. Fifty-two percent reported that they had eaten one or more fresh fruits on the previous day*; we found that 39 percent ate whole fruit on the recall day. In the California survey, 42 percent reported eating green salad and 67 percent reported eating one or more vegetables on the recall day. In our data, 37 percent had a salad and 43 percent had at least one serving of a garden vegetable on the recall day.

Dietary guidelines have emphasized the importance of a balanced and varied diet. Diets that include either no servings or very few servings of fruit and vegetables lack both balance and variety. We found that almost 50 percent of the population had at most a single serving of a vegetable; even among those who had two servings of a vegetable on the recall day, 21 percent had two servings of the same vegetable (other than a garden vegetable). For fruit and fruit juice, about three-fourths had at most a single serving.

Studies that have assessed health benefits from vegetable consumption found them primarily for garden vegetables.[24-26] We found that less than half the population had a garden vegetable on the recall day.

As in the paper on food choices,[1] we found that consumption of fruit and vegetables (with the exception of garden vegetables) was lower among Blacks than Whites, even when income was taken into account (data available on request to authors). Not only did fewer Blacks than Whites meet the guidelines, but larger proportions had at most a single serving on the recall day.

Consumption of the recommended five servings of fruit and vegetables is consistent with a nutritionally adequate diet as well as with the health recommendations cited above. For example, diets including multiple servings of these foods provide substantial amounts of dietary fiber and vitamins A and C. While caloric intake increases with the numbers of servings of both fruit and vegetables, this increase is not associated with greater obesity. As more servings of vegetables are eaten, more grams of fat are consumed, but the P/S ratio also rises.

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*Data provided by S. B. Foerster, personal communication.

The fact that fruit and vegetables are important sources of dietary fiber may not be well understood by the general public. In our data, a diet that included five servings of fruit and vegetables provided approximately 17 grams of fiber; at this level, the addition of two slices of whole grain bread would bring the total to 20 or more grams. Nutrition education campaigns might well emphasize that consumption of the recommended five-a-day is important in achieving the dietary fiber goals, as well as in increasing micronutrient intake.

Evidence has been accumulating on the relation of fruit and vegetable consumption to health, as is reflected in the NAS recommendations to increase consumption of these foods. Numerous studies suggest that we can help prevent several types of cancer by increasing our fruit and vegetable consumption.[24-26] However, the discrepancy between the America diet and the fruit and vegetable guidelines, as shown in a one day recall, is so enormous as to call into question the societal commitment to these guidelines. The public is either unaware of the likely benefits of including these foods in the daily diet or, although aware, is unwilling or cannot afford to follow the guidelines.

Fruit and vegetable consumption has never been the object of a national campaign. A recent editorial in this Journal called for "the most vigorous, collaborative nutrition campaign ever launched"[27] to make the public aware of the connection between food intake and health outcome. Any such campaign should take into account the fruits and vegetables most frequently eaten, encouraging increased consumption of those already relatively popular, and targeting demographic groups where consumption is particularly low. In addition to public education, societal commitment might include actions to improve the affordability of many fruits and vegetables and to increase the numbers of servings obtainable through school lunches, meals on wheels, and other food programs, as well as industry actions to promote increased consumption of these foods.

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REFERENCES

1. Patterson BH, Block G: Food choices and the cancer guidelines. Am J Public Health 1988; 78:282–286.
2. US Department of Health and Human Services, Public Health Service: The Surgeon General's Report on Nutrition and Health. DHHS (PHS) Pub. No. 88-50210. Washington, DC: Govt Printing Office, 1988.
3. Committee on Diet and Health, Food and Nutrition Board, Commission on Life Sciences, National Research Council: Diet and Health: Implications for Reducing Chronic Disease Risk. Washington, DC: National Academy Press, 1989.
4. US Department of Agriculture, US Department of Health and Human Services: Nutrition and Your Health: Dietary Guidelines for Americans. Home and Garden Bulletin No. 232. Washington, DC: Govt Printing Office, 1980.
5. Cronin FJ, Shaw AM, Krebs-Smith SM, Marsland PM, Light L: Developing a food guidance system to implement the dietary guidelines. J Nutr Educ 1987; 19:281–302.
6. National Center for Health Statistics: Plan and Operation of the Second National Health and Nutrition Examination Survey, 1976–1980. Vital and Health Statistics Series 1, No. 15. DHEW Pub. No. (PHS) 81-1317. Washington, DC: Govt Printing Office, 1981.
7. National Center for Health Statistics: Dietary Intake Source Data: United States, 1976–1980. Vital and Health Statistics, Series 11, No. 231. DHHS Pub. No. 83-1681. Washington, DC: Govt Printing Office, 1983.
8. Shah BV: SESUDAAN: Standard Errors Program for Computing of Standardized Rates from Sample Survey Data. Research Triangle Park, NC: Research Triangle Institute, 1981.
9. Dresser CM: From nutrient data to a data base for a health and nutrition examination survey. Organization, coding, and values—real or imputed. Proceedings of the Eighth National Data Base Conference, Minneapolis, MN, July 1983.
10. Block G, Dresser CM, Hartman AM, Carroll MD: Nutrient sources in the American diet: Quantitative data from the NHANES II survey. Am J Epidemiol 1985; 122:13–40.
11. National Center for Health Statistics: Public Use Data Tape Documentation—Total Nutrient Intake, Food Frequency, and Other Related Dietary Data Tapes. Tape number 5701. National Health and Nutrition Examination Survey, 1976–1980. US Dept of Health and Human Services, Public Health Service, Office of Health Research, Statistics, and Technology. Hyattsville, MD: NCHS, February 1982.
12. US Department of Agriculture, Agricultural Research Service: Nutritive Value of American Foods, in Common Units. Agriculture Handbook No. 456. Washington, DC: Govt Printing Office, 1975.
13. National Cancer Institute: Diet, Nutrition and Cancer Prevention: A Guide to Food Choices. US Dept of Health and Human Services, NIH Pub. No. 85-2711. Washington, DC: Govt Printing Office, 1985.
14. US Department of Agriculture, Agricultural Research Service: Dietary Levels of Households in the U.S., Spring 1965. Preliminary Report ARS 62-17. Washington, DC: Consumer and Food Economics Research Division, Agricultural Research Service, US Dept of Agriculture, 1968.
15. American Heart Association: Nutrition Committee Rationale for Diet-Heart Statement of the American Heart Association, Dallas, TX. Circulation 1982; 65:839A–854A.
16. Select Committee on Nutrition and Human Needs, US Senate: Dietary Goals for the United States. Washington, DC: Govt Printing Office, 1977.
17. US Department of Agriculture and US Department of Health, Education, and Welfare: Food is More Than Just Something to Eat. US Department of Agriculture Home and Garden Bulletin No. 216, 1976.
18. US Department of Agriculture, Science and Education Administration: Food and Nutrient Intakes of Individuals in 1 Day in the United States, Spring 1977. NFCS 1977–78 Preliminary Rpt No. 2, 1980.
19. US Department of Agriculture, Human Nutrition Information Service: Nationwide Food Consumption Survey, Continuing Survey of Food Intakes by Individuals, Men 19–50 Years, 1 Day, 1985. NFCS, CSFII Report No. 85-3. Washington DC: USDA, 1986.
20. US Department of Agriculture, Human Nutrition Information Service: Nationwide Food Consumption Survey, Continuing Survey of Food Intakes by Individuals, Women 19–50 Years and Their Children 1–5 Years, 1 Day, 1985. NFCS, CSFII Report No. 85-1. Washington DC: USDA, 1985.
21. US Department of Agriculture, Human Nutrition Information Service: Nationwide Food Consumption Survey, Continuing Survey of Food Intakes by Individuals, Women 19–50 years and their children 1–5 years, 1 Day, 1986. NFCS, CSFII Report No. 86-1. Washington, DC: USDA, 1987.
22. Popkin BM, Haines PS, Reidy KC: Food consumption trends of US women: Patterns and determinants between 1977 and 1985. Am J Clin Nutr 1989; 49:1307–1319.
23. Abrams B, Kristal A, DiSogra L, Ho EE, Foerster S, Chan BS, Parker L: How well do Californians meet dietary guidelines for cancer risk reduction? 116th Program of the Annual Meeting of the American Public Health Association 1988 (Abstract).
24. Micozzi MS: Foods, micronutrients, and reduction of cancer. *In:* Moon TE, Micozzi MS (eds): Nutrition and Cancer Prevention: Investigating the Role of Micronutrients: New York: Marcel Dekker, 1989; 213–242.
25. Peto R, Doll R, Buckley J, Sporn M: Can dietary beta-carotene materially reduce human cancer rates? Nature 1981; 290:201–208.
26. Block G: Ascorbic acid in cancer prevention: The epidemiologic evidence. Am J Clin Nutr 1990; 52: (in press).
27. Crawford P: The nutrition connection: Why doesn't the public know? (editorial) Am J Public Health 1988; 78:1147–1148.

APPENDIX

We define K indicator (dummy) variables for the K + 1 cells in our classification (K = 15 for the 4 x 4 cross-classification of numbers of servings of fruits and vegetables.) The remaining P - K variables, $X_{K+1}, \ldots, X_p$, represent indicator variables for the categories into which the adjustment variables have been divided (in our case, the three age, two race, and two sex categories generate four additional dummy variables). The continuous variable for which we are adjusting is denoted Y_k, $k = 1, \ldots, K$ (for example, Y_k might be number of calories consumed by those in the k^{th} cell). Then the adjusted mean for the k^{th} cell, $k = 1, \ldots, K$, is given by the following regression model:

$$\overline{Y}_k = b_0 + b_k + b_{K+1}\overline{X}_{K+1} + \ldots + b_P\overline{X}_P$$

and the adjusted mean for the (K + 1)th cell is given by

$$\overline{Y}_{K+1} = b_0 + b_{K+1}\overline{X}_{K+1} + \ldots + b_P\overline{X}_P$$

where $\overline{X}_{K+1} \ldots \overline{X}_P$ are means for the whole sample, or, in our case, simply the proportion (in the population) in each age, sex, or race category. A simple interpretation can be given to the adjusted means. The adjusted mean for the kth cell is the mean that would have been generated by the data if the population in the kth cell had the same distribution (with respect to the race, sex, and age categories) as the entire population.

The estimated variance of the adjusted mean can be found by using the estimated variances and covariances of the b's, obtained from the SURREGR procedure,[8] software that takes into account the complex sampling design and the sampling weights.

Executive Women and Health: Perceptions and Practices

JUDITH H. LAROSA, PHD, RN

Abstract: The purpose of this study was to obtain a socioeconomic/health profile of a select group of executive women, to understand more about their personal and professional lives, and to examine how these factors relate to their overall health. The data were obtained from a self-administered 73-item questionnaire that was mailed during spring 1987 to the 1,000 members of a professional executive women's organization with 15 chapters across the United States. Findings suggest that the women in executive positions do not necessarily compromise their health. In comparison with a group of age/gender/education matched working women, the overall wellness and risk assessment scores were remarkably similar. The study group, however, reported greater life satisfaction, stronger social support, and excellent health status; the overwhelming majority was satisfied with their personal and professional lives and believed that they were in control of both. This perception, coupled with the relatively high wellness scores, suggests that on average this group of executives may be in better health than had been predicted as women rose to executive positions within organizations. (*Am J Public Health* 1990; 80:1450–1454.)

Introduction

The role of women has changed—both inside and outside the home. Since the beginning of the twentieth century, many women have moved from the traditional rural culture of child-rearing and farm work to the modern urban culture of nannies and executive boardrooms.

Although the move from farm to city and from home to office has been evolving since the early 1900s, it was during World War II that the major and more permanent change occurred. In 1890, women constituted 18 percent of labor force participation, by 1945 that number had risen to 36 percent, and in 1989, women represented 54 percent of the labor force participation.[1,2]

During World War II, as throughout history, women took on the work responsibilities vacated by men who left home and jobs to fight wars. Unlike the aftermath of previous wars, however, American women did not follow historical precedent and return home to their traditional responsibilities. They continued to work outside the home and over time began to take on occupations previously held by men—medicine, the law, engineering, politics, and others. Gradually they began to take on the power and responsibilities associated with these occupations. In many cases, women added these outside responsibilities to the ones they already had at home—managing the children and the household.

Yet, little attention has been focused on the impact of these changes. Studies are only just now emerging that begin to examine the complex nature of the home/work/health relationships on working women. Some investigators have suggested that functioning in different roles poses health problems for working women,[3–5] while others have suggested that it is beneficial.[6–8] Some have suggested that a critical factor in these relationships is the perception that the woman holds about her life situation.

Prior health studies focused primarily on women in blue-collar occupations, or on women in white-collar occupations who worked at relatively low to mid-level positions.[3–24] Existing studies of executive women usually do not include health data.[25,26] Indeed, only three studies were identified that focused on the health of women in executive positions—either in very large companies or in the federal government.[27–29] The purpose of this study, therefore, was to obtain a socioeconomic/health profile of a select group of executive women, to understand more about their personal and professional lives, and to examine how these factors relate to their overall health.

Methods

The data were obtained from a self-administered questionnaire that was mailed during spring 1987 to the 1,000 members of a professional executive women's organization with 15 chapters across the United States. Entry into the organization is by invitation only and is based on criteria that insists on each member having "substantial influence in her field . . . regardless of title." The organization criteria also states that "there are many influential and powerful women outside large organizations, thus the size of the organization should not be considered as an important criterion." Occupations include chief executive officers, lawyers and judges, physicians and scientists, administrators, politicians, the clergy, artists, and writers. The majority, 54.8 percent, listed their title as executive. The mean income of the respondents was $133,000 per annum; median income was $96,000. The income distribution was positively skewed. The income levels ranged from no income for a Roman catholic nun who was a hospital administrator to several chief executive officers who reported over $3 million in salary per annum. Because of the wide range of incomes, further analyses of income against other key variables are underway.

The questionnaire consisted of 73 items in four major categories—sociodemographic, general health, job satisfaction, and home/work/health relationships. Sociodemographic items were based on those used in the National Health and Nutrition Examination Surveys and National Health Interview Surveys.[30,31] Participants were also asked job-related questions and queried about who bears the major responsibility for making major financial decisions and arranging child care.

The general health section consisted of the 1984 32-item* Centers for Disease Control Health Risk Appraisal (CDC HRA) with the exception of one item reflecting exposure to

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Address reprint requests to Judith H. LaRosa, PhD, RN, Executive Women's Health Study, 1958 Dundee Road, Rockville, MD 20850. Dr. LaRosa is also with the Department of Health Education, University of Maryland, College Park. This paper, submitted to the Journal February 20, 1990, was revised and accepted for publication April 6, 1990.

*One question was dropped from the original questionnaire at the request of the organization's board. The question was considered offensive and board members felt that such a question would seriously impair the willingness of members to respond. The excluded question was "How many of the following do you usually do? Hitch-hike or pick up hitch-hikers? Carry a gun or knife for protection? Keep a gun at home for protection? Criticize or argue with strangers? Live or work in a high crime area? Seek entertainment at night in high-crime areas or bars?

violence.[32] This instrument was used for two reasons. In addition to providing an assessment of the individual's perception of her health status, the CDC HRA also provided a limited assessment of the respondent's level of personal satisfaction—an important psychosocial variable previously shown to be related to the development of disease.[9-11]

The job satisfaction section consisted of two scales developed by Karasek and colleagues—a five-item job satisfaction scale and a nine-item decision latitude scale.[33-37] These variables were added because professional satisfaction and perception of job mastery are believed to be related to health.[4,9-11,33-37]

Perceptions about control over their lives and the impact of their personal and professional lives on physical health and mental well-being were examined using five questions developed by the investigator. Finally, two open-ended questions were included at the end of the questionnaire for the respondents to add comments on their personal and professional lives and their health, and to reflect on the study. Analyses of the responses to these questions are not included in this paper; they will be the subject of a future analysis.

After a three-phase mailing, designed to maximize the response rate, 66 percent (660/1,000) of the members had responded. Of those 660, 590 cases (59 percent of the total) were complete and usable; 545 of the cases represented executives who were employed full-time. They are the focus of this paper.

Complete data for comparing respondents and nonrespondents were available on only two factors: education and marital status. No differences were found between the two groups on these factors. While differences may have existed, other demographic factors such as age, number of years married, spouse education and income, level of personal and professional satisfaction, and the entry criteria for membership in the organization suggest that this particular population of executive women is fairly homogeneous. The assumption that the respondents and nonrespondents are similar seems reasonable.

Results

The 545 respondents were predominantly White, middle-aged, well-educated, well-paid executives. The majority were married and had an average of two children. Almost 19 percent of the children were 18 years of age or younger. The majority of the spouses, like their wives, were well-educated executives. Table 1 summarizes the selected demographic findings and job characteristics.

Although the majority of the couples had equally well-paying, executive positions, the distribution of jobs at home was not as equitable. While the responsibility for making major financial decisions was a shared responsibility between the partners, the responsibility for arranging child care usually fell to the woman. Seventy-three percent of the respondents reported arranging child care 75 percent or more of the time.

Over half the respondents have had at least two job changes in the past 10 years, usually representing an advancement. While the majority of the respondents worked for a firm other than their own, 28 percent owned their own company. The majority reported high levels of job satisfaction (median equaled 90 on a scale of 0 to 100) and decision latitude (median equaled 86 on a scale of 0 to 100)—indicating an ability to make autonomous decisions regarding their work direction and pace.

TABLE 1—Profile of 545 Full-Time Women Executives

Factors		
Age (years) (Mean/Median)	49.7/48.0 years	
Race (% White)	90.1	
Education (% ≥ Masters)	57.0	
Spouses' Education (% ≥ Masters)	66.1	
Married (%)	59.5	
Married (years)	20.0	
Children (% with)	73.0	
Children (Average Number)	2.0	
Children (% with ≤ 18 years of age)	19.0	
Annual Personal Income (Mean/Median)	$133,785/$96,000	
Spouse's Annual Personal Income (Mean/Median)	$143,102/$90,000	
Annual Total Family Income (Mean/Median)	$271,401.00/$175,000.00	
Hours worked per week (Mean/Median)	54.0/54.0	
Self-employed (% who owned firm)	27.5	
Occupation	Respondent	Spouse
Business Executive	54.8	45.7
Lawyer	11.2	17.2
Physician	0.0	6.2
Government Official	9.0	0.0
College Administrator/Professor	6.2	4.2
Other	18.8	26.0

NOTE: Other includes editor, publisher, author/correspondent, physician, CPA, architect, psychologist, performer/artist

Over 56 percent of the respondents believed that they had a great deal of control over their personal and professional life. An additional 31 and 34 percent in each category (personal and professional, respectively) believed they had a moderate amount of control. In addition, the majority of respondents (77 percent) reported being at least mostly satisfied with their lives.

Respondents furthermore reported that their personal and professional lives had a strongly positive effect on both physical health and mental well-being. In both personal and professional categories, however, the positive effect on mental well-being was stronger than the positive effect on physical health (data available from author upon request).

In general, respondents believed that they were in good health and reported maintaining positive health practices. The mean CDC HRA wellness score, based on a 100-point scale, was 86, with a median score of 87.

This score requires a reference point to indicate where this group falls on the 100-point continuum in comparison with other groups. Although no entirely appropriate comparison group who had completed the CDC HRA could be found for these senior-level executive women, their responses were compared with those from a matched group of working women in the Fitness Research Center data base at the University of Michigan.** Each of the participants in the study group (N = 533)*** was matched on age and education with 20 working women from the Fitness Research Center data base for a total of 10,660. Because neither of the two groups is necessarily representative of all working women nor all executive women, the findings should be interpreted with caution.

**The Fitness Research Center at the University of Michigan maintains a current data base of over 100,000 health risk appraisals collected from the University of Michigan Business School and from diverse data bases provided by investigators from across the United States.

***Twelve cases of the original 545 were not used either because of insufficient data or because respondents were over 70 years of age. Thus, the 533 used represented complete cases.

TABLE 2—Demographics, Disease Risk Factors, and Health Practices and Perceptions among the Study Group and the Matched Group

Factors	Study Group (N = 533*)	Matched Group (N = 10,660)
Sociodemographics		
Age (Mean)	49	49
Marital Status (%)		
Married	58.5	65.3
Single	12.2	10.5
Divorced	20.1	14.1
Separated	2.6	2.2
Widowed	5.3	7.4
Race/Ethnic Group (%)		
White	89.9	89.9
Black	7.1	5.2
Hispanic	1.9	1.2
Asian	0.6	2.5
Native American	0.0	0.9
Other	0.6	0.2
Education (%)		
≤ High School	7.1	7.1
Some College	2.1	2.1
College or Professional	90.6	90.6
Risk Factors		
Height/Weight (Mean)		
Height (Inches)	65.5	65.5
Weight (Pounds)	136.7	137.4
Smoking (%)		
Smoker	12.0	17.7
Ex-smoker	38.3	23.3
Never Smoker	49.7	58.9
Among Smokers		
Average/day	11.4	19.0
Among Ex-smokers		
Years Stopped	5.4	10.3
Blood Pressure (%)		
High SBP** ≥ 140 mm Hg	5.5	21.9
High DBP** ≥ 90 mm Hg	0.4	17.2
Mean SBP (mm Hg)	117.8	120.1
Mean DBP (mm Hg)	75.0	77.1
Know Blood Pressure	44.5	68.5
Blood Cholesterol (%)		
BC ≥ 5.7 mmol/L	25.6	36.1
Mean Total BC (mmol/L)	5.0	5.6
Know Blood Cholesterol	16.1	25.0
Alcohol Use (%)		
Drinker	72.2	45.3
Ex-drinker	2.1	2.3
Never Drinker	25.7	52.5
Among Drinkers Only (%)		
Light (≤ 7/week)	57.4	72.5
Medium (8–24/week)	39.2	26.9
Heavy (≥ 25/week)	3.1	0.6
Average/week	8.1	7.8
Drug Use		
Non (Rarely) Use	92.3	93.5
Light (Occasional)	5.6	4.8
Heavy (Almost daily)	2.1	1.6
Health Practices		
Physical Activity		
Regular (≥ 3 times/week)	49.3	44.1
Occasional	37.1	43.6
Little	13.5	12.3
Sleep		
≤ 6 hours/night	24.6	27.1
7–8 hours/night	72.6	72.0
≥ 9 hours/night	2.8	1.7
Pap Smear		
Annually	68.3	63.5
Every 3 Years	22.0	22.9
< Every 3 Years	7.1	9.7
Almost Never	0.2	0.7
Breast Self-examination		
Monthly	21.2	36.7
Every Few Months	40.3	35.5
Rarely/Never	38.5	27.8
Annual Rectal Examination	47.1	37.1

Table 2 summarizes the CDC HRA health findings from the study reported here and compares them with the CDC HRA health findings from the matched reference group.

The average wellness scores and the risk assessment scores between the two groups were remarkably similar. Yet, further examination reveals several important differences. Fewer of the study group were married than were the matched group with the major difference occurring among those who were divorced. Fewer of the study group smoked than did the matched group, yet far more of the study group reported drinking than did the matched group. Although the number who reported heavy drinking (≥25 drinks of alcohol per week) was small, the study group had over three times as many heavy drinkers as the matched group.

Blood pressure and total blood cholesterol levels were reported as slightly lower among the study group than the matched group.† However, this should be interpreted carefully as the study group of executives was far less knowledgeable about their blood pressure and blood cholesterol values than the matched group. The majority of the executives in the study group did not know either value.

Differences existed between the two groups concerning regular exercise as well as miles driven and seat belt use. The study group exercised more regularly (49 percent) than did the matched group (44 percent). The executives drove, on average, considerably more miles per year than did the matched group and were far more likely to use seat belts (80 percent) than did the matched group (36 percent).

Discussion

This study provides some preliminary data that suggest that the women in executive positions do not necessarily compromise their health. Indeed, this particular group of executive women perceived themselves to be quite healthy, which was supported by their overall wellness and risk assessment scores. In comparisons with a group of age/gender/education-matched working women, the overall wellness and risk assessment scores were remarkably similar. The study group reported greater life satisfaction, stronger social support, and excellent health status. The overwhelming majority of the study group were satisfied with their personal and professional lives and believed that they were in control of both. This perception, coupled with the relatively high wellness scores, suggests that this group of executives may be in better health than had been anticipated as women rose to executive positions within organizations.

The greater numbers of those who drank and who drank heavily, as well as the greater number of divorcees among the study group, requires further examination. Perhaps among a sub-set of these women, there are those for whom the executive lifestyle had a negative effect.

That the executives drink more is not entirely unexpected. Although fewer business lunches and dinners today are conducted with alcohol as an important social ingredient, alcohol is still consumed at such meetings. It may be that alcohol consumption is perceived as a necessary social part of doing business. Or, it may be that these executives really do consume alcohol more often than do other types of working women, possibly as a way of reducing stress. Yet, other behaviors that have been linked to stress reduction,

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†Where blood pressure and blood cholesterol data are missing, the CDC HRA program automatically assigns an age/gender/race mean to that case.

TABLE 2—Continued

Factors	Study Group (N = 533*)	Matched Group (N = 10,660)
Seatbelt Use		
Regular (≥ 75%)	80.5	36.4
Semi-regular (<75%)	6.0	37.4
Non-user	13.5	26.1
Average Use (%)	82.9	47.4
Average driving miles/years	26,538	11,191
Perceptions		
Life Satisfaction		
Completely	77.3	60.4
Quite	21.0	28.5
Somewhat	1.3	9.9
Not Very	0.4	1.2
Perceived Social Support		
Strong	69.6	52.7
Average	21.2	40.5
Below Average	4.1	5.9
Unsure	2.1	1.1
Perceived Health Status		
Excellent	61.4	36.0
Good	34.0	55.8
Fair	3.9	7.6
Poor	0.8	0.8
Risk Assessment		
Average Risk Age (years)		
Actual	49.6	48.6
Appraised	48.1	47.4
Achievable	46.0	44.9
Projected Death Rate All Cause Death/1,000 Persons during Next 10 Years		
Actual	77.4	75.1
Appraised	68.3	71.6
Achievable	57.7	55.4
Wellness Score (Mean)	85.5	78.3

*533 represents cases which are complete.
**SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure

such as smoking and overeating, appear to be less common with this group of executives compared to the matched group. In fact, more positive stress-reducing activities, such as regular exercise, appear to be practiced by almost half of the executive group.

It is not surprising that at the time of the study (1987) many of the participants and matched group were unaware of their blood cholesterol levels. The National Cholesterol Education Program had just been initiated and related public education was in its infancy. What is surprising is the number of executives who did not know their blood pressure values. The National High Blood Pressure Education Program and its public education efforts have been strong in the United States for the past 17 years. Knowledge about one's "number" and awareness about the disease has been increasing since the inception of this national educational effort. Indeed, knowledge about blood pressure and action to control it has been highly correlated with education and income, especially among females. The reason for this lack of knowledge about blood cholesterol and blood pressure levels is puzzling and is worthy of further investigation.

The greater effort expended on exercise is certainly within keeping among men with higher education. Now Matthews, *et al*, have reported similar findings among women with college and advanced degrees, e.g., the higher the education, the greater the energy expenditure.[38]

While this study may begin to dispel some of those notions of compromised health among women executives, it raises far more questions than it answers. Clearly the study has limitations. It captures only one moment in time and it does not represent all executive women. However, it provides suggestions that might direct further study. The study and the review of the literature argue vigorously for more investigation in this particular area of executive working women, and in the larger concern of the interrelationships of home, work, and health for all working women.

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REFERENCES

1. US Bureau of the Census: Historical statistics of the United States, Colonial times to 1970. Bicentennial Edition, Part 1. Washington, DC: The Bureau, 1975; 131–132.
2. US Department of Labor: Employment in perspective: Women in the labor force. Bureau of Labor Statistics, Report 782:1–3, Fourth Quarter 1989.
3. Zimmerman MK, Hartley WS: High blood pressure among employed women: A multi-factor discriminant analysis. J Health Soc Behav 1982; 23:205–220.
4. Haynes SG, Feinleib M: Women, work, and coronary heart disease. Am J Public Health 1980; 70:133–140.
5. Waldron I: The coronary-prone behavior pattern, blood pressure, employment, and socioeconomic status in women. J Psychosom Res 1978; 22:79–87.
6. Kandel DB, Davies M, Ravies VH: The stressfulness of daily social roles for women: marital, occupational, and household roles. J Health Soc Behav 1985; 26:64–78.
7. Verbrugge LM: Women's social roles and health. Women: A Developmental Perspective. Department of Health and Human Services, NIH Pub. No. 82-2298. Washington, DC: Govt Printing Office, 1978; 48–77.
8. Chassie MB, Bhagat RS: Role stress in working women: Differential effect on selected organizational outcomes. Groups and Organization Studies 1980; 5:224–233.
9. Karasek R, Gardell B, Lindell J: Work and non-work correlates of illness and behavior in male and female Swedish white collar workers. J Occup Behav 1987; 8:187–207.
10. Kalberg AL: Work values and job rewards: A theory of job satisfaction. Am Soc Rev 1977; 42:124–143.
11. Verbrugge LM: Work satisfaction and physical health: J Community Health 1982; 7:262–283.
12. Celentano DD, MacQueen DV: Alcohol consumption patterns among women in Baltimore. J Stud Alcohol 1984; 45:355–358.
13. Chernik DA, Phelan JG: Attitudes of women in management. I. Job satisfaction: A study of perceived need satisfaction as a function of job level. Int J Soc Psychiatry 1974; 20(1–2):94–98.
14. Cox T, Thirlaway M, Cox S: Occupational well-being: Sex differences at work. Ergonomics 1984; 27(5):499–510.
15. Hazuda HP, Hafner SM, Stern M, Knapp JA, Eifler CW, Rosenthal M: Employment status and women's protection against coronary heart disease. Am J Epidemiol 1986; 123:623–640.
16. Hauenstein LS, Kasl SV, Harburg E: Work status, work satisfaction, and blood pressure among married Black and White women. Psychol Women Q 1977; 1:334–349.
17. Haw M: Women, work, and stress: A review and agenda for the future. J Health Soc Behav 1985; 26:132–143.
18. Hibbard JH, Pope CR: Employment status, employment characteristics, and women's health. Women and Health 1985; 10:59–77.
19. Jenkins R: Minor psychiatric morbidity in employed young men and women and its contribution to sickness absence. Br J Ind Med 1985; 42:147–154.
20. Kelly KE, Houston BK: Type-A behavior in employed women: Relation to work, marital, and leisure variables, social support, stress, tension, and health. J Personality Soc Psychol 1985; 48:1067–1079.
21. Stellman SD, Stellman JM: Women's occupation, smoking, cancer, and other diseases. Ca-A Cancer Journal 1981; 31:29–43.
22. Waldron I, Herold J, Dunn D, Staum R: Reciprocal effects of health and labor force participation among women: Evidence from two longitudinal studies. JOM 1982; 24:126–132.
23. Wheeler AP, Lee ES, Loe HD: Employment, sense of well-being, and use of professional services among women. Am J Public Health 1983; 73:908–911.

24. Woods NF, Hulka BS: Symptom reports and illness behavior among employed women and homemakers. J Community Health 1979; 5:36–42.
25. Hymowitz C, Schellhardt TD: The glass ceiling. *In:* A special report: The corporate women. Wall Street Journal March 24, 1986.
26. von Glinow MA, Mercer AK: No room at the top: The systematic exclusion of women from leadership of corporate America. Unpublished study from the University of Southern California at Los Angeles, Department of Industry, School of Business, 1987.
27. Detre K, Feinleib M, Matthews KA, Kerr BW: The federal women's study. *In:* Eaker ED, Packard B, Wenger NK, Clarkson TB, Tyroler HA (eds): Proc of the Conference on Coronary Heart Disease in Women. New York: Haymarket Doyma Incorporated, 1987; 78–82.
28. Cooper CL, Melhuish A: Executive stress and health: Differences between men and women. JOM 1984; 26:99–104.
29. Davidson MJ, Cooper CL: Occupational stress in female managers: A comparative study. J Management Stud 1984; 21:185–205.
30. National Center for Health Statistics: Plan and Operation of the Second National Health and Nutrition Examination Survey, 1976–1980. Vital and Health Statistics, Series 1, No. 15. DHHS Pub. No. PHS 81-1317. Washington, DC: Govt Printing Office, July 1981.
31. National Center for Health Statistics: Current Estimates From the National Health Interview Survey: United States, 1985. Vital and Health Statistics, Series 10, No. 160. DHHS Pub. No. PHS 86-1588. Washington, DC: Govt Printing Office, September 1986.
32. Centers for Disease Control; CDC health risk appraisal user's manual and interim update supplement. Atlanta: Division of Health Education, Center for Health Promotion and Education, 1984.
33. Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T: Job decision latitude, job demands, and cardiovascular disease: A prospective study of Swedish men. Am J Public Health 1981; 71:694–705.
34. Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL: Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). Am J Public Health 1988; 78:910–918.
35. Schwartz JE, Pieper CF, Karasek RA: A procedure for linking psychosocial job characteristic data to health surveys. Am J Public Health 1988; 78:904–909.
36. Reed DM, LaCroix AZ, Karasek RA, Miller D, Maclean CA: Occupational strain in the incidence of coronary heart disease. Am J Epidemiol 1989; 129:495–502.
37. Pieper CF, LaCroix AZ, Karasek RA: The relation of psychosocial dimensions of work with coronary heart disease risk factors: A meta analysis of 5 US data bases. Am J Epidemiol 1989; 129:483–494.
38. Matthews KA, Kelsey SF, Meilahn EN, Kuller LH, Wing, RR: Educational attainment and behavioral and biologic risk factors for coronary heart disease in middle-aged women. Am J Epidemiol 1989; 129:1132–1144.

Innovations in State and Local Government Awards Program Invites 1991 Applications

The Innovations in State and Local Government Program has issued a call for applications for its 1991 Innovations Awards Program to recognize creative government efforts at the state, county, municipal, special authority, and tribal levels. Ten winners, selected by a national committee of leaders from government, education, business, and the media, will receive grants of up to $100,000 each by the Ford Foundation.

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To be considered, all applications must be received no later than 5:00 p.m., Wednesday, January 9, 1991.

Day Care Characteristics Associated with *Haemophilus influenzae* Disease

JAY D. WENGER, MD, LEE H. HARRISON, MD, ALLEN HIGHTOWER, MS, CLAIRE V. BROOME, MD,
Haemophilus influenzae STUDY GROUP*

Abstract: To identify characteristics of day care facilities associated with *H. influenzae* disease, we compared 92 licensed facilities in which a case of *H. influenzae* disease had occurred with randomly selected facilities at which no cases occurred. Matched univariate analysis showed that personnel at facilities where *H. influenzae* disease occurred were more likely than those at control facilities to use towels or handkerchiefs to wipe children's noses, admit children who were not toilet trained or had diarrhea ("liberal fecal policy"), had a narrower age range, were more likely than control facilities to be for-profit and less likely to use volunteers. In a multivariate model that adjusted for age range, profit status and liberal fecal policy, towel or handkerchief use (OR 5.5, 95% CI: 1.1, 30) was the only variable independently associated with case facilities. This is the first association of a specific day care practice with *H. influenzae* disease. (*Am J Public Health* 1990; 80:1455–1458.)

Introduction

Whereas 20 years ago most children were cared for at home, today most women with children less than 6 years of age are working outside the home, and most of their children receive care at day care facilities.[1,2] Numerous studies have suggested that children attending day care centers were more likely than those cared for at home to suffer from a variety of infectious diseases.[3–9] In particular, attendance at day care facilities is a major risk factor for primary invasive *Haemophilus influenzae* disease.[10–12] Since nearly all *H. influenzae* disease is primary (i.e., is not a result of transmission from another known case), we performed a case-control study comparing licensed day care facilities where a case of *H. influenzae* disease occurred with licensed day care facilities which did not have cases of disease in the same period.

Methods

Surveillance for Invasive *H. influenzae* Disease

Laboratory-based active surveillance for invasive *H. influenzae* disease was performed in four states (Tennessee, Missouri, New Jersey, and Washington) and Los Angeles County, California from January 1, 1986 to December 31, 1986. The details of this system have been previously reported.[13] A contact in each acute care hospital in the area was responsible for reporting to the surveillance coordinator all cases of *H. influenzae* disease from that hospital. A child was considered to be a case of *H. influenzae* disease if *H. influenzae* was cultured from a normally sterile site. When the project ended, surveillance coordinators reviewed ICD-9 discharge diagnosis codes in each hospital to evaluate the sensitivity of the surveillance system.

Selection of Case Day Care Facilities

A child was considered a day care facility attendee if he/she was being cared for along with at least one other unrelated child for at least four hours during the week before becoming ill with *H. influenzae*. The surveillance coordinator contacted the parents of all children between the ages of 1 and 60 months by telephone to determine day care facility attendance. A day care facility was entered into the study only if it appeared on the list of licensed day care facilities provided by the state or county health departments, since no listing of unlicensed facilities existed to select controls for cases attending unlicensed facilities. The director of each case day care facility was contacted by telephone and, after explanation of the study, offered the opportunity to participate. If a case day care facility refused to participate, the case was eliminated from the study. If the director of the case day care facility agreed to cooperate, the surveillance coordinator obtained the case center's characteristics via questionnaire at that time.

Selection of Control Day Care Facilities

For each enrolled case day care facility a size-weighted, randomly ordered list of licensed day care facilities in the surveillance area was generated. The first day care facility on the list was chosen as a control day care facility if it:

- matched the case day care facility for type, i.e. day care home (in a residence) or nonresidential day care center;
- was not a case day care facility itself; and
- had not already been selected previously as a control.

The director of each potential control day care facility was contacted by telephone until a day care facility was found that would have cared for children in an age range including the age of the case at the time of case admission and was willing to participate.

Statistical Methods

Univariate and multivariate models were evaluated using matched conditional logistic regression.[14] Analyses performed on subsets of the data (for example, for cases under two years of age or for day care homes vs day care centers) contained only sets where both the case and control met the

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From the Meningitis and Special Pathogens Branch and Statistical Services Activity, Center for Infectious Diseases, CDC; and the *Haemophilus influenzae* Study Group. Address reprint requests to Jay D. Wenger, MD, Building 1, Room 5409, CO9, Centers for Disease Control, Atlanta, GA 30333. This paper, submitted to the Journal December 11, 1989, was revised and accepted for publication May 22, 1990.

*The *Haemophilus influenzae* Study Group: Sandra L. Sitze and Margaret Spurrier (Missouri Department of Health, Jefferson City, MO); Robert C. McCready, David R. Cundiff, and Maureen C. Farrell (New Jersey Department of Health, Trenton, NJ); Janice D. Harwell (Washington State Department of Social and Health Services, Seattle, WA); Lori Chronis, Betty Grimes, Carolyn C. Hoppe, and Stephen H. Waterman (Los Angeles County Department of Health Services, Los Angeles, CA); Brenda K. Boner, Rose A. Kelley, Lewis B. Lefkowitz, Jr, and Jo A. Taylor (Department of Health and Environment, State of Tennessee; Department of Preventive Medicine, Vanderbilt University School of Medicine, Nashville, TN); and Suzanne Gaventa and Arthur L. Reingold (CDC, Atlanta, GA).

criteria. Multivariate modeling was hypothesis-oriented and did not use stepwise variable selection methods.

Results

Surveillance and Identification of Case/Control Pairs

During 1986, 1,230 cases of invasive *H. influenzae* disease in children less than or equal to 60 months of age were reported from the five surveillance areas. ICD-9 discharge diagnosis code reviews were available for all surveillance areas except New Jersey. Of 408 cases reported by discharge diagnosis, 370 (90 percent) were detected by active surveillance. Day care status was determined for 1,081 (88 percent) of the 1,227 cases. A total of 358 cases attended day care facilities. Of these, 198 (57 percent) attended unlicensed day care facilities, and further information was not available for another 26 (7 percent), due to parental unwillingness to identify the facility. Parents or day care facility directors of 14 of the remaining 134 cases refused to participate. An additional four cases were deleted from the analysis because they occurred in facilities already enrolled as cases in the study, and we did not want to represent the same case facility twice in the analysis. These four facilities were not clearly different from other case facilities. Case and control pairs were obtained for 92 of the remaining 116 eligible cases (79 percent). The 24 eligible cases for whom controls were not available did not differ from the 92 included in the study by age, sex, race, or clinical syndrome, nor did their facilities differ by type or size or any of the variables associated significantly with case facilities in the univariate analysis. Sixty-nine case and control pairs were non-residential facilities (day care centers) and 23 pairs were residential facilities (day care homes). There were no significant differences in age, sex, race, or disease syndrome between day care attendees who were enrolled in the study and those who were not enrolled (Table 1).

Risk Factors

Univariate Analysis—Table 2 shows odds ratios of potential risk factors for *H. influenzae* disease in the analysis of all day care facilities regardless of age of case, enrollment, or type of facility. Separate analyses stratifying for age of case (less than or equal to 2 years of age, or older than 2 years of age), enrollment (less than or equal to 20 or greater than 20 children/day care facility) and type of day care facility (day care home vs day care center) did not identify other risk factors except as noted below.

TABLE 1—Characteristics of Day Care-Associated *H. influenzae* Cases

Characteristics	Cases Enrolled in Case-Control Study n = 92 (%)	Cases Not Enrolled[*] n = 266 (%)
Sex		
Male	49 (53)	130 (49)
Age (mo)		
0–6	13 (14)	50 (19)
7–11	25 (27)	71 (27)
12–23	29 (31)	83 (32)
24–35	10 (11)	37 (14)
36–59	15 (16)	25 (9)
Race		
White	63 (69)	181 (70)
Black	19 (21)	46 (18)
Asian/South Pacific	1 (1)	4 (2)
Hispanic	1 (1)	17 (7)
Unknown	8 (9)	18 (7)
Clinical syndrome (cultures positive)		
Cerebrospinal fluid (CSF) only	30 (33)	79 (30)
CSF + blood	29 (32)	94 (35)
Blood only	33 (35)	91 (34)

Note: Percent in parentheses.

[*] Because of unlicensed or unknown licensure status of facility, or refusal by parents or day care facility.

Age Range—The mean age range of children cared for in case-day care facilities was 67 months, while that in control centers was 83 months ($p = 0.023$). This difference was primarily a function of the difference in the mean age of the oldest child in the day care facility, since the mean age of the youngest child did not differ significantly between case and control day care facilities (due partly to the matching design).

Exclusion Policies—As shown in Table 3, control day care facilities were more likely to exclude children with diarrhea than were case day care facilities (OR 0.35, 95% CI: 0.14, 0.90). The two variables measuring exposure to feces were combined to form a third variable—liberal fecal policy (accepting non-toilet-trained children (Table 1) or not excluding children with diarrhea). The univariate odds ratio for liberal fecal policy was 3.3 (95% CI: 1.3, 8.3).

Business Status—Failure to use volunteers and for-profit status were highly associated variables with 15 percent of for-profit facilities using volunteers, compared with 39 percent of non-profit facilities ($p < 0.001$). Not using volunteers had an odds ratio of 2.6 and for-profit status had an odds ratio of 1.4.

Other Factors—Several other factors evaluated are listed in Table 2. Of note, use of a towel or handkerchief to wipe childrens' noses was associated with case day care facilities with an unadjusted odds ratio of 4.5.

Multivariate Analysis—A multivariate model was used to evaluate the significance of potential risk factors which were hypothesized to be related to disease and which, in the univariate analysis, showed either elevated (or reduced) odds ratios or associations ($p < .05$) with case facilities. Since two pairs of variables were closely related (age range/age of oldest child, and profit status/use of volunteers), only one of each pair was used in the multivariate analysis. The results of a multivariate model constructed with the variables of age range, profit status, liberal fecal policy and towel or handkerchief use are summarized in Table 4. Towel or handkerchief use is the most strongly associated variable (OR 5.5, 95% CI = 1.01, 30).

Discussion

During the course of this study, we found, as have others,[15] that many day care facility characteristics are closely associated. The matching technique and the association of many day care facility characteristics with the matching variables may have decreased our ability to identify risk factors in this study.

Use of towels or handkerchiefs to wipe childrens' noses was the most important risk factor for occurrence of *H. influenzae* disease in our multivariate model. This practice may facilitate transfer of the pathogen from child to child, since spread of *H. influenzae* organisms occurs through exchange of oropharyngeal secretions. The towel-handkerchief variable may represent a direct means of person-to-person spread of *H. influenzae*. *H. influenzae* organisms adhere to human nasopharyngeal mucous.[16] Murphy, *et al*, recently found that nasal secretions of *H. influenzae* carriers had much higher concentrations of *H. influenzae* than cough or oropharyngeal secretions, and that *H. influenzae* could

TABLE 2—Risk of *H. influenzae* disease for Selected Characteristics of Day Care Facilities, Univariate matched analysis (unadjusted)

Characteristics	n[*] (sets)	Cases	Controls	OR	95% CI
Mean age range (years)	91	5.1	6.4	0.9	0.82, 0.98
Mean age of oldest child in day care facility (years)	91	6.2	7.6	0.9	0.82, 0.98
Mean age of youngest child in day care facility (months)	91	7.8	8.9	0.98	0.94, 1.02
Mean hours open	89	58	58	1.0	0.87, 1.14
Mean number enrolled	92	70	64	1.0	1.00, 1.01
Mean number of staff	91	11	10	1.02	0.98, 1.06
Mean tuition ($/week, full time)	85	55	52	1.01	0.99, 1.02
Mean # children in class	82	9.7	9.8	1.0	0.93, 1.07
Mean # children in class 0–23 months old	65	5.5	5.3	1.04	0.91, 1.19
Mean age range in class	89	24	27	.99	0.97, 1.01
Mean age of youngest in class	89	13	13	.99	.94, 1.05
Mean age of oldest in class	89	37	40	.99	0.97, 1.01
% run for profit vs. nonprofit	92	63	57	1.37	0.72, 2.62
% using volunteers	88	16	34	0.38	0.19, 0.80
% accepting children for 1 day	92	33	48	0.54	0.29, 1.00
% accepting children not toilet trained	82	90	83	2.0	0.75, 5.33
% using disposable tissues	91	90	95	0.5	0.15, 1.66
% using towels or handkerchiefs[**]	83	10.9	2.4	4.5	0.97, 21

[*] analysis performed only on sets for which specific data were available.
[**] all facilities using towels or handkerchiefs also reported using disposable tissues.

TABLE 3—Exclusion Policies of Case and Control Day Care Facilities

Policy	n sets	case (%)	control (%)	odds ratio	95% CI
No policy	89	6.7	4.5	1.7	0.4, 7.0
Exclude fevers	92	86	90	0.64	0.25, 1.64
Exclude runny nose	90	10	10	1.0	0.29, 3.45
Exclude cough	89	12	19	0.54	0.21, 1.35
Exclude diarrhea	89	75	88	0.35	0.14, 0.90
Exclude vomiting	89	91	97	0.38	0.10, 1.4

TABLE 4—Multivariate Model (82 sets)

	OR	95% CI
Age range	.90	.80, 1.01
Profit (vs non-profit)	2.1	.93, 4.9
Towel/handkerchief use	5.5	1.01, 30
Liberal fecal policy	2.3	0.78, 6.9

survive for up to 48 hours on fomites such as water-absorbent towels.[17] Our study provides evidence that the route of transmission hypothesized by the laboratory studies of Murphy, *et al*, is important in day care facilities.

Several organizational characteristics were associated with case day care facilities in the univariate analysis. Case day care facilities were more likely to have a narrower age range and younger age of eldest child than control day care facilities. The age of youngest child in the day care facility did not differ. When day care centers were analyzed separately, these risk factors were still present. The same trends were present for day care homes, but were less stable. Although the association of narrower age range with risk of disease may be related to having more children of a younger age (and therefore at higher risk of *H. influenzae* disease) in a case day care facility, we could not evaluate this possibility since information on age of every child in each day care facility was not collected. However, neither number of classrooms nor number of children <23 months of age in each classroom differed between case and control day care facilities.

Slightly more control day care facilities than case day care facilities advised or required vaccination for *H. influenzae*, but the difference was minimal (42–44 percent vs 34–40 percent, depending on age of the child). It is unlikely that this difference accounted for lack of disease in control day care facilities because of the small size of the difference, and because more than two-thirds of all cases occurred in children too young to receive the vaccine (less than 18 months of age). The trend toward a more active vaccination policy, however, may represent better awareness of the disease and means of prevention.

Liberal fecal policy (evaluated because colonization with some enteric bacteria with cross-reacting antigens may be important in *H. influenzae* disease),[18] profit status, and age range were less strongly associated with case day care facilities. These variables were all related and may be surrogates for other more specific risk factors. For instance, profit status was also associated with hepatitis A incidence in the study of day care facilities in Arizona.[15] Although several other day care facility characteristics are likely to be associated with whether the facility is run as a for-profit or non-profit organization, it was not clear (from either this or the previous analysis of hepatitis A in day care facilities) how this could relate causally to incidence of *H. influenzae* disease.

Although a role for use of towels or handkerchiefs to wipe noses in transmission of infectious diseases has been suggested previously, this is the first study to identify it as a risk factor in the day care setting. Additional measures must be taken to eliminate this practice. Several other variables less strongly associated with risk of disease included liberal fecal policy, profit status, use of volunteers, and age range. They may be surrogates for more specific risk factors which remain to be identified.

REFERENCES

1. Bureau of Labor Statistics, US Department of Labor: News Release 87-343. Washington, DC: Department of Labor, August 1987.

2. US Bureau of the Census: Current Population Reports Series p-70, No. 9, Who's minding the kids? Child care arrangements: Winter 1984–85. Washington, DC: Bureau of the Census, 1987.
3. Johansen AS, Leibowitz A, Waite LJ: Child care and childrens' illness. Am J Public Health 1988; 78:1175–1177.
4. Wald ER, Dashevsky B, Byers C, Buerra N, Taylor F: Frequency and severity of infections in day care. J Pediatr 1988; 112:540–546.
5. Anderson LJ, Parker RA, Strikas RA, *et al*: Day care center attendance and hospitalization for lower respiratory tract illness. Pediatrics 1988; 82:300–308.
6. Bartlett AV, Moore M, Gary GW, Starko KM, Erben JJ, Merideth BA: Diarrheal illness among infants and toddlers in day care centers. II. Comparison with day care homes and households. J Pediatr 1985; 107:503–509.
7. Fleming DW, Cochi SL, Hightower AW, Broome CV: Childhood upper respiratory tract infections: To what degree is incidence affected by day care attendance? Pediatrics 1987; 79:55–60.
8. Doyle AB: Incidence of illness in early group and family day care. Pediatrics 1976; 58:607–613.
9. Strangert K: Respiratory illness in preschool children with different forms of day care. Pediatrics 1976; 57:191–196.
10. Redmond SR, Pichichero ME: *Haemophilus influenzae* type b disease: An epidemiologic study with special reference to day care centers. JAMA 1984; 252:2581–2584.
11. Istre GR, Conner JS, Broome CV, Hightower A, Hopkins RS: Risk factors for primary invasive *Haemophilus influenzae*: Increased risk from day care attendance and school-aged household member. J Pediatr 1985; 106:190–195.
12. Cochi SL, Fleming DW, Hightower AW, *et al*: Primary invasive *Haemophilus influenzae* type b disease: A population-based assessment of risk factors. J Pediatr 1986; 108:887–896.
13. Harrison LH, Broome CV, Hightower AW, *et al*: A day care based study of the efficacy of *Haemophilus* b polysaccharide vaccine. JAMA 1988; 260:1413–1418.
14. Breslow NE, Day NE: Statistical methods in cancer research. Lyons, France, International Agency for Research on Cancer, 1980; Vol 1: 162–189.
15. Hadler SC, Erben JJ, Francis DP, Webster HM, Maynard JE: Risk factors for Hepatitis A in day care centers. J Infect Dis 1982; 145:255–261.
16. Farley MM, Stephens DS, Mulks MH, *et al*: Pathogenesis of IgA1 protease-producing and -nonproducing *Haemophilus influenzae* in human nasopharyngeal organ cultures. J Infect Dis 1986; 154:752–759.
17. Murphy TV, Clements JF, Petroni M, Conry S, Stetler L: *Haemophilus influenzae* type b in respiratory secretions. Pediatr Infect Dis J 1989; 8:148–151.
18. Schneerson R, Robbins JB: Induction of serum *H. influenzae* type b capsular antibodies in adult volunteers fed cross-reacting *E. coli* 075:K1OO:H5. N Engl J Med 1975; 292:1093–1096.

NIH Consensus Panel Issues Report on IVIG

Earlier this year, the National Institute of Allergy and Infectious Diseases and the Office of Medical Applications of Research of the National Institutes of Health convened a Consensus Development Conference on *Intravenous Immunoglobin: Prevention and Treatment of Disease*.

The consensus panel has now issued its report containing conclusions and recommendations from this conference.

Single copies of the report may be obtained from: William H. Hall, Director of Communications, Office of Medical Applications of Research, NIH, Bldg. 1, Rm 259, Bethesda, MD 20892; (301) 496-1142.

OMAR will also provide multiple copies of a two-page summary statement of the conference findings to organizations that wish to distribute copies to its members.

Injury and Disability in Matched Men's and Women's Intercollegiate Sports

RICHARD R. LANESE, PHD, RICHARD H. STRAUSS, MD, DANIEL J. LEIZMAN, MD, AND ANN MARIE ROTONDI, MA

Abstract: Eight matched men's and women's intercollegiate varsity teams were studied prospectively for one academic year to determine the incidence of athletic injury and resulting disability. Sports in which both men and women participated in a comparable manner were chosen: basketball, fencing, gymnastics, swimming, tennis, indoor track, outdoor track, and volleyball. Men (232) and women (150) were injured at comparable rates, 42 percent versus 39 percent. When adjusted for exposure time, seven of the eight sports continued to show similar injury rates. Women gymnasts, however, experienced .82 injuries per 100 person-hours of exposure as compared to .21 injuries for the men ($p = .0001$). Disability was greater in women gymnasts, 7.44 days per 100 person-hours versus 1.15 days for men ($p = .0004$). Percent of season lost to injury was also greater for women gymnasts. Types and sites of injury were similar for men and women, with sprains and strains accounting for over half of all injuries. We found no evidence for gender differences in matched sports except for gymnastics, in which technically diverse events may have accounted for the differences observed. (*Am J Public Health* 1990; 80:1459–1462).

Introduction

Women's participation in intercollegiate athletics has increased dramatically in recent years. Several studies comparing injury rates in men and women participating in similar sports have reported conflicting results.[1–3] Some studies have noted higher injury rates in female gymnasts and basketball players.[4–7] However, few studies have been conducted on mature men and women.[8–10]

In view of the conflicting results of previous studies and the paucity of information on mature athletes, the present study was undertaken to provide injury data on men and women engaged in similar sports while controlling for the length of time each athlete was exposed to injury. Earlier studies have rarely controlled for exposure time. If gender differences are observed, a larger study may be designed to investigate possible risk factors such as technical differences within a sport or biological differences associated with injury.

Methods

The study was conducted as a prospective cohort study for the period of one academic year at a large university. Eight matched men's and women's varsity sports were studied. These were sports in which both men and women participated in a comparable manner and included basketball, fencing, gymnastics, swimming, tennis, indoor track, outdoor track, and volleyball.

One of the authors (A.M.R.) visited each team weekly to record the appropriate injury information for the week. Injury data came from the medical records kept by the team physicians and by certified and student athletic trainers.

An athletic trainer or student trainer was present at each team practice and competition and was responsible for collecting the injury and disability data. Injuries not attributable to the team sport, as well as all illnesses, were excluded from the study.

We defined an injury as a traumatic medical problem due to sports participation and resulting in loss of time from practice or competition. When an injury occurred during practice or competition and the athlete was unable to complete the day's activity, one-half day of disability was assigned. One whole day of disability was assigned for each full day of practice or competition lost. On weekends, teams usually had only one day off. For example, if an athlete was injured on Saturday, had no scheduled practice on Sunday, and returned to practice on Monday, half of Sunday was assigned as injury time off. Disability days were calculated for each athlete.

We studied each team from the first official day of practice until the last day of regular competition or practice. Hours of practice and competition were estimated by either direct observation or interviews with coaches and athletes, or a combination of both methods. This provided indices independent of differences in exposure time. Exposure time was calculated for each athlete and for each sport by subtracting disability days as well as other absences from the total time available for practice and competition.

To compare injuries between men and women, we utilized the following indices: 1) injuries per 100 persons; 2) injuries per 100 person-hours of exposure; 3) disability days per 100 person-hours of exposure; 4) percent of season lost to injury; and 5) percent of persons injured. These indices were computed for each athlete and each team. Because injuries were not normally distributed and, more than likely, not independent, the nonparametric Wilcoxon Rank Sum statistic,[11] corrected for ties, was employed to test gender differences within each sport and for all sports after assigning an injury rate to each athlete. This procedure was used for two indices: injuries per 100 person-hours of exposure, and percent of season lost to injury. The Mantel-Haenszel statistic[12] provided a test of difference in the proportion of men and women injured over all sports together. The relative risk of injury and its 95% confidence interval was estimated for males using the direct precision-based method.[13]

If the true proportions of male and female athletes who are injured are in the range of 40 percent to 60 percent, the present study sample has approximately an 80 percent chance of detecting a 15 percent difference when alpha is at .05.

Types of injury and anatomical sites were also determined. No practicable statistical methods of analyses were available to evaluate gender differences for these parameters.

Results

The study cohort was composed of 232 men and 150 women. Table 1 shows the frequency and percent injured for

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From the Department of Preventive Medicine, Ohio State University College of Medicine, Columbus. Address reprint requests to Richard R. Lanese, PhD, Professor of Preventive Medicine, Ohio State University, 320 West 10th Avenue, Columbus, OH 43210. Dr. Strauss is Associate Professor of Preventive Medicine and Internal Medicine, and team physician; Dr. Leizman was a medical student and Ms. Rotondi was a graduate student, all at Ohio State University, Columbus. This paper, submitted to the Journal September 5, 1989, was revised and accepted for publication April 17, 1990.

TABLE 1—Injuries among Male and Female Athletes in Eight Matched Intercollegiate Sports during One Year at a Large University

Sport	Gender	Persons on Team	Persons Injured (%)	Number of Injuries	Injuries per 100 Persons
Basketball	M	12	10 (83)	18	150
	F	14	11 (79)	19	136
Fencing	M	18	5 (28)	5	28
	F	6	3 (50)	3	50
Gymnastics	M	24	23 (96)	52	217
	F	11	11 (100)	30	273
Swimming	M	36	14 (39)	21	58
	F	21	8 (38)	8	38
Tennis	M	12	5 (42)	6	50
	F	11	3 (27)	4	36
Track, Indoor	M	57	21 (37)	28	49
	F	37	12 (32)	16	43
Track, Outdoor	M	56	9 (16)	11	20
	F	37	4 (11)	4	11
Volleyball	M	17	11 (65)	13	76
	F	13	6 (46)	7	54
All Sports	M	232	98 (42)*	154	66
	F	150	58 (39)	91	61

*Relative risk = 1.25 (CI 0.88, 1.43).

each of the eight matched sports. While the percentage of athletes injured varied considerably between sports, within sports there appear to be only small and inconsistent gender differences. For all sports combined, where 42 percent of the men and 39 percent of the women were injured, the Mantel-Haenszel χ^2 was 0.53 ($p \approx .50$). The relative risk, adjusted for between sport differences, was 1.25 with a 95% confidence interval of 0.88 to 1.43.

The rate of injury expressed as injuries per 100 persons was similar for all sports combined, 66 injuries per 100 persons for men, and 61 per 100 persons for women. Both men and women participating in gymnastics appear to have higher rates of injury than athletes in the other sports. All but one of the 35 gymnasts reported at least one disabling injury during the season. Women gymnasts were injured at a somewhat higher rate than men—273 versus 217 per 100 persons, respectively. When the incidence of injuries is expressed in terms of exposure time, women gymnasts sustained 0.82 injuries per 100 person-hours of exposure, while men incurred only 0.21 injuries (Table 2). This difference was significant by the Wilcoxon Rank Sum Test ($p = .0001$). There were no gender differences observed for the seven other sports nor for all sports combined.

Table 2 also summarizes disability days for men and women by sport. Women gymnasts had the highest rate of disability, 7.44 days per 100 person-hours of exposure, while men gymnasts had only 1.15 disability days, a sixfold difference ($p = .0004$). We were unable to detect gender differences in any of the remaining seven sports and, more importantly, when all eight sports were combined. For all sports, both men and women missed 1.4 days for every 100 hours of exposure, a period approximated by five to six weeks of practice and competition.

The types and sites of injury occurring during the period of study appear to be similar for both men and women. Strains and sprains were clearly the most common types of injury (Figure 1), accounting for over half of all injuries for both men and women.

The most frequent site of injury was foot and ankle (Figure 2). There appears to be little difference between sexes with respect to site. However, on closer inspection, injuries to the upper extremity (shoulder, upper arm, hand, wrist, elbow, and forearm) occurred more frequently in men (27 percent of injuries) than in women (13 percent of injuries).

Discussion

We relied on the medical records as kept by the team physicians and athletic trainers. The data were reviewed and accumulated once each week by one of the authors. We assumed that the reporting of injuries would be equally reliable across all sports and gender.

The present study involved 382 men and women over a period of one year at a single university. Thus, the question of repeatability of the findings from year to year, as well as the generalizability of the findings from one setting to another, may be questioned. While the total number of athletes studied was fairly large, the number of men and

TABLE 2—Injury and Disability Rates among Male and Female Athletes in Eight Matched Intercollegiate Sports during One Year at a Large University

Sport	Gender	Mean Hours of Exposure*	Injuries per 100 Person-Hours	(p)**	Disability Days	Disability Days per 100 Person-Hours	(p)**
Basketball	M	337	0.45	(.90)	65.5	1.62	(.48)
	F	283	0.48		69.0	1.74	
Fencing	M	278	0.10	(.27)	21.5	0.43	(.47)
	F	273	0.18		3.5	0.21	
Gymnastics	M	1020	0.21	(.0001)	281.5	1.15	(.0004)
	F	365	0.82		274.0	7.44	
Swimming	M	469	0.12	(.67)	241.5	1.43	(.77)
	F	488	0.08		63.5	0.62	
Tennis	M	305	0.16	(.37)	88.5	2.42	(.37)
	F	375	0.10		23.5	0.57	
Track, Indoor	M	266	0.18	(.44)	326.0	2.15	(.33)
	F	281	0.15		51.0	0.49	
Track, Outdoor	M	151	0.13	(.56)	143.5	1.70	(.52)
	F	92	0.12		71.5	2.11	
Volleyball	M	408	0.19	(.93)	52.0	0.75	(.48)
	F	353	0.15		22.5	0.49	
All Sports	M	397	0.18	p = .99	152.5	1.44	p = .49
	F	305	0.21		72.3	1.38	

*adjusted for missed practice and competition
**probability of difference between groups by Wilcoxon Rank Sum test

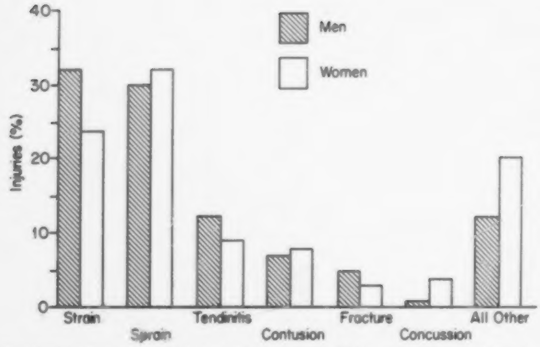

FIGURE 1—Percent of Injuries by Type among Male and Female Athletes in Eight Matched Intercollegiate Sports during One Year at a Large University

women in each paired sport was relatively small, resulting in low statistical power for detecting sport-specific gender differences. While gender differences in gymnastics were sufficiently large to overcome this problem, there may be differences in other sports that we were unable to detect.

Our estimates of exposure time for each athlete are crude, but should be useful when comparing gender differences for a single sport. For example, in basketball, men and women play the game in a similar manner and probably have comparable periods of activity and inactivity. To the extent that this is true, any gender difference in observed rate will be free of bias from exposure difference in a given sport.

We expected modest differences in exposure times between women and men in the same sport. However, we did not anticipate the size of the differences observed. For the 16 teams, exposure times ranged from 92 hours for women's outdoor track to 1,020 hours for men's gymnastics. It is clear from Table 2 that within-sport and between-sport comparisons of injury must take these exposure differences into account. Gymnastics reflects the greatest exposure disparity between women and men, 365 hours and 1,020 hours, respectively. Yet, women report a higher incidence of injuries per 100 persons.

We found little difference between men and women in terms of injury or disability in the eight sports except for gymnastics. Other investigators[2,10,14] as well, have reported differences in injury rates and types of injury in male and female gymnasts, with females showing somewhat higher rates. The differences that we observed are largely a result of expressing injuries in terms of exposure time. Only small differences between the sexes were observed when injuries were expressed per 100 persons or as percent of persons injured (Table 1).

When we expressed disability as the percent of season lost for each athlete, the median for women gymnasts, 8.21 percent, was significantly higher than the median for men, 3.15 percent ($p < .01$). No other team lost as high a percentage of their season due to disability. Ten of the 14 teams had medians at zero percent.

It is generally accepted that men gymnasts use predominantly upper body musculature and women use predominantly lower body musculature. This is consistent with the upper extremity injury difference noted earlier. The injury and disability differences that have been reported in this paper and elsewhere may be more a consequence of the different types of events and apparatus than of gender. Women gymnasts participate in four events and men in six. Only two of the events utilize the same apparatus: floor exercise and vault.

It is interesting to note that, while women and men gymnasts comprised only 9 percent of the athletes followed during the year of study, they accounted for almost 34 percent of all injuries. The apparent excess of injuries in gymnasts may be a result of the special requirements of the sport. Injuries that might be regarded as inconsequential and not reported by athletes in other sports such as football may impair the performance of the gymnast.

The main finding of this paper suggests that, when men and women compete in sports that are technically similar, the

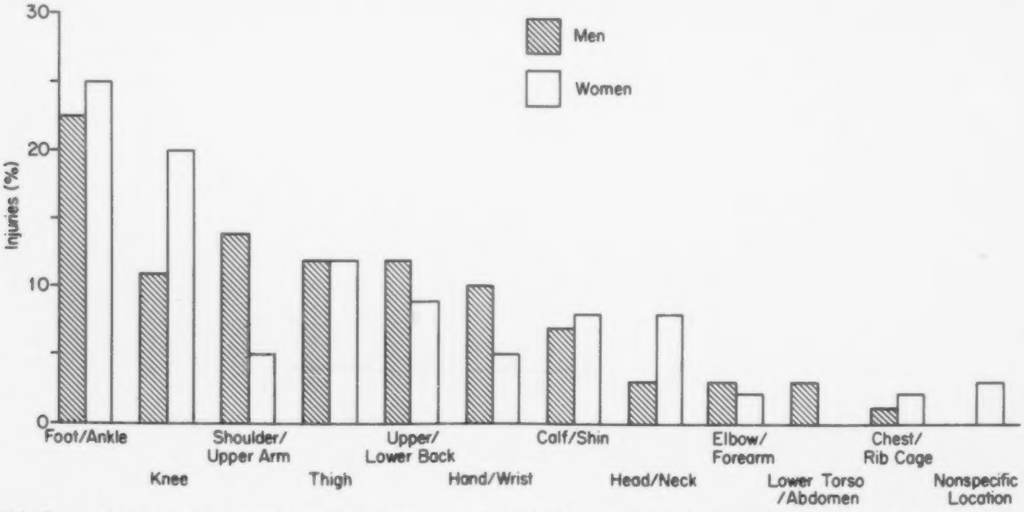

FIGURE 2—Percent of Injuries by Site among Male and Female Athletes in Eight Matched Intercollegiate Sports during One Year at a Large University

rates of injury are approximately equal. When they compete in sports that are technically different, there is little reason to expect injury rates to be the same. This may account for our single positive finding—that men and women gymnasts differed in injury rates.

ACKNOWLEDGMENT

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REFERENCES

1. Cox JS, Lenz HW: Women midshipmen in sports. Am J Sports Med 1984; 12:241–243.
2. Garrick JG, Requa R: Medical care and injury surveillance in the high school setting. Phys Sportsmed 1981; 9:115–120.
3. Shively RA, Grana WA, Ellis D: High school sports injuries. Phys Sportsmed 1981; 9:46–50.
4. Lowry CB, Leveau BF: A retrospective study of gymnastics injuries to competitors and noncompetitors in private clubs. Am J Sports Med 1982; 10:237–239.
5. Moretz A, Grana WA: High school injuries. Phys Sportsmed 1978; 6:92–95.
6. Chandy TA, Grana WA: Secondary school athletic injury in boys and girls: a three year comparison. Phys Sportsmed 1985; 13:106–111.
7. Zelisko JA, Noble HB, Porter M: A comparison of men's and women's professional basketball injuries. Am J Sports Med 1982; 10:297–299.
8. Whiteside PA: Men's and women's injuries in comparable sports. Phys Sportsmed 1980; 8:130–140.
9. Haycock CE, Gillette JV: Susceptibility of women athletes to injury. JAMA 1976; 236:163–165.
10. Clarke KS, Buckley WE: Women's injuries in collegiate sports. Am J Sports Med 1980; 8:187–191.
11. Wilcoxon Rank Sum Test—Sokal RR, Rohlf FJ: Biometry Second Edition. New York: W.H. Freeman and Company, 1981.
12. Mantel Haenszel Test—Schlesselman JJ: Case-Control Studies. New York: Oxford University Press, 1982.
13. Relative Risk and its 95% CI—Kleinbaum DG, Kupper LL, Morganstern H: Epidemiologic Research. Belmont, CA: Lifetime Learning, 1982.
14. Garrick JG, Requa RK: Injuries in high school sports. Pediatrics 1978; 61:465–469.

National Conference on Cancer Nursing: Call For Abstracts

Nursing professionals are invited to submit abstracts for poster presentations for the American Cancer Society's Sixth National Conference on Cancer Nursing. The conference, "From Primary to Tertiary Care: Cancer Across the Life Span," will be held July 25–27 in Seattle, Washington.

Within the theme, abstract topics may be related to clinical practice, education, research, and nursing administration. The deadline for abstract submission is February 1. For additional information, contact: Terri Ades, RN, Coordinator for Nursing Programs, ACS National Office, 1599 Clifton Rd, NE, Atlanta, GA 30329; (404) 329-7655.

This conference is the sixth in a series of national nursing conferences which are held every four years, and whose purpose is to improve the quality of cancer care provided to individuals and their families. The conference program is directed at nurse generalists, primary care nurses, oncology nurses, and other members of the health profession whose practice is devoted to providing care to individuals and their families throughout the life span. The conference is sponsored by the American Cancer Society, in cooperation with the American Nurses' Association Council on Primary Health Care Nurse Practitioners, Association of Pediatric Oncology Nurses, and the Oncology Nursing Society.

Coding External Causes of Injury (E-Codes) in Maryland Hospital Discharges 1979–88: A Statewide Study to Explore the Uncoded Population

BARUCH MARGANITT, MD, MPH, ELLEN J. MACKENZIE, PHD, GORDON S. SMITH, MBCHB, MPH, AND ANNE M. DAMIANO, SCM

Abstract: We examined the trends in hospital discharge E-coding in Maryland over a 10-year period. The overall proportion of E-coded discharges has increased from 40 percent in 1979 to 55 percent in 1988. E-coding was lower in the severely injured, the elderly, and patients with long hospital stays. Our findings demonstrate that E-code reporting varies because of the limited number of data fields available for coding of discharge diagnoses. Universal, systematic reporting of E-codes in hospital discharge data is essential if these data are to provide critically needed information about nonfatal injuries. Hospital discharge data formats should contain separate fields for E-codes and the use of these codes, we believe, should be mandated. (*Am J Public Health* 1990; 80:1463–1466).

Introduction

Efforts to examine trends in the incidence of injury, patterns of acute care, and patient outcomes have generally required substantial investments in case ascertainment and data collection. The increasing availability of computerized statewide hospital discharge data offer new opportunities for timely and efficient data collection and policy analysis of issues related to the prevention of injuries and trauma care delivery.[1–8] The recent development of a computerized conversion table that translates ICD-9CM coded discharge diagnoses into Abbreviated Injury Scale (AIS) scores has led to new applications of these databases, providing population-based data specific to the severity of injuries.[8–11] Currently, 28 states require that uniform hospital discharge data be collected on all acute care hospitalizations.[8]

The lack of consistent coding of the external cause of injury limits the usefulness of these databases.[12,13] Although a classification of external causes exists within the structure of the International Classification of Diseases (ICD E-codes-),[17] hospitals vary in their use of these codes. Accurate data on the causes of nonfatal injuries and a means to measure their trends are not available in the United States. Underreporting of E-codes in hospital discharge abstracts has been recognized as a major limitation in their use for these purposes.[12–15]

To better understand factors responsible for incomplete E-coding, we analyzed 10 years of Maryland hospital discharge data. The specific objectives of the study were: to examine time trends in the level of E-coding in the statewide population-based hospital discharge data; and to determine demographic and clinical characteristics of the uncoded population (the missing E-code population), in order to identify strategies for improving reporting of E-codes.

Methods

Uniform hospital discharge abstract data for the state of Maryland during the 10 years from 1979 to 1988 were used to examine trends in E-coding over time. All 52 non-federal acute care hospitals in Maryland are mandated by law to submit uniform data to the State's Health Services Cost Review Commission (HSCRC). External causes of injury are reported as ICD-9CM E-Codes and can be included in the database as one or more of five possible discharge diagnoses.

The Study Population consisted of the 349,487 hospital discharges in the state of Maryland with a principal diagnosis of traumatic injury. Traumatic injuries were defined as those injuries coded with ICM-9CM diagnoses codes 800–959 excluding: late effects (905–909), foreign bodies (930–939), and early complications (958). Poisonings (960–979), toxic effects (980–989), other and unspecified effects of external causes (990–995), and complications of medical and surgical care (996–999) were also excluded. E-coding status of each discharge abstract was defined as one of the following three categories based on existence of an E-code and availability of ICD-9CM code recording space: 1) E-coded discharge, 2) Uncoded discharge with no coding space available (no E-code, not codable), 3) Uncoded discharge with coding space available (no E-code, codable). Using the 1988 data, coding status was examined by age of the patient, injury severity, body region of the injury assigned as the principal diagnosis (head-neck/extremities), length of hospital stay, and level of hospital care, i.e.: 1) Specialty Referral Trauma Centers (SRTC) (One adult and one pediatric); 2) Regional Trauma Center (RTC) (nine centers); 3) Community Hospital (Com Hosp) (41 hospitals). Injury Severity was assessed by the Injury Severity Score (ISS)[16] and was assigned to each discharge using the ICDMAP software[9,10] that maps ICD-9CM coded diagnoses into Abbreviated Injury Scale (AIS) scores and ISS. E-code status was examined for the 10 years 1979–1988.

Results

The proportion of E-coded trauma discharges increased over the study period by 38 percent, from 40 percent in 1979 to 55 percent in 1988 (Figure 1). Over the same period, however, the proportion of uncoded discharges that had vacant space available for additional codes (no E-code, codable) decreased from 98 percent in 1979 to 53 percent in 1988.

Increasing age was associated with a decrease in the percent of E-coded discharges as well as an increase in the proportion of not codable discharges (Figure 2). In 1988, the year with the highest coding rate, 73 percent of the 0–5 age

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From the Health Services Research and Development Center and Injury Prevention Center, Johns Hopkins School of Hygiene and Public Health. Address reprint requests to Baruch Marganitt, MD, MPH, Health Services Research and Development Center, Johns Hopkins School of Hygiene and Public Health, 624 N. Broadway Street, Baltimore, MD 21205. This paper, submitted to the Journal January 16, 1990, was revised and accepted for publication April 20, 1990.

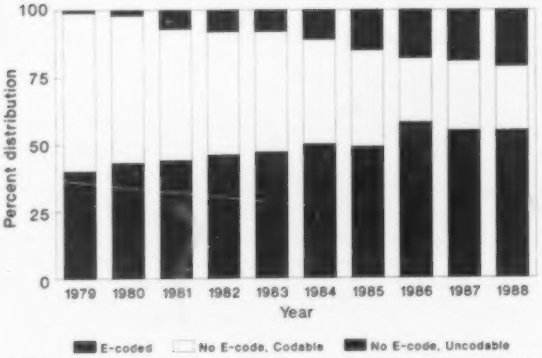

FIGURE 1—E-Coding Status by Year, Maryland Trauma Discharges, 1979–88

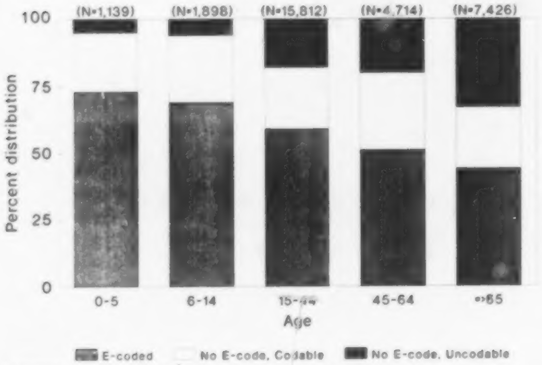

FIGURE 2—E-Coding Status by Age, Maryland Trauma Discharges, 1988

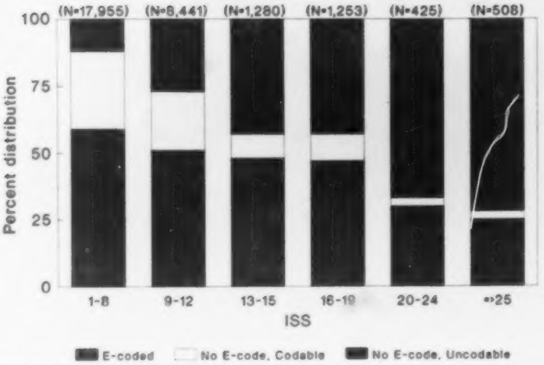

FIGURE 3—E-Coding Status by Injury Severity Score, Maryland Trauma Discharges, 1988

group discharges were E-coded as compared to 44 percent in the ≥65 age group. However, in age group 0–5, 18 percent of the uncoded discharges were not codable, as compared to 59 percent in age group ≥65. This differs from earlier study years; in 1979, for example, the proportion of E-coded discharges ranged from 44 percent in 0–5 age group to 40 percent in ≥65, while the percent of uncoded discharges that were not codable ranged from 0–1 percent.

As the Injury Severity Score (ISS) increased the proportion of E-coded discharges decreased and the proportion of uncoded discharges that were not codable increased (Figure 3). In 1988, the proportion of E-coded discharges ranged from 59 percent of ISS group 1–8 to 25 percent of ISS ≥25. However, in ISS group 1–8, 29 percent of the uncoded discharges were not codable, as compared to 92 percent of the uncoded discharges of ISS group ≥25. In the early years of the study period, E-coding status did not vary with ISS; for example, in 1979, the proportion of E-coded discharges ranged from 42 percent of ISS 1–8 to 39 percent of ISS ≥25; out of the uncoded discharges the percent of not codable discharges, however, was very low ranging from 0–2 percent.

Table 1 compares the 1988 E-coding status within ISS subgroups between the two most prevalent body-region categories: head/neck and extremities. In both groups the proportion of E-coded discharges decreased with increasing ISS. However, the proportion of coded discharges in the extremities group was lower than the head-neck group in all ISS categories, with more notable differences in the most severe groups (ISS 16 and higher). There were also marked differences between the head/neck and extremities groups in the proportion of uncoded discharges that were not codable. For example, in the ISS 16–19 Head/Neck group 47 percent of the discharges were uncoded, and 72 percent of them were not codable. This is compared to discharges with a principal injury to the extremities of which 76 percent were uncoded, and of them 95 percent were not codable.

In 1988, 64 percent of the 1 day LOS group were E-coded, as compared to 25 percent of the 29–56 days, and 12 percent of LOS ≥57 days (Figure 4). Of the uncoded discharges the proportion that were not codable increased from 17 percent of the uncoded discharges in the 0–1 day LOS group to 96 percent of the uncoded discharges in the ≥57 days LOS group.

E-coding increased over the study period in all levels of care, however trauma centers coded a higher proportion of discharges. For example, in 1988 over 60 percent of trauma center discharges were E-coded as compared to 47 percent of the discharges from community hospitals (Figure 5). In community hospitals, 70 percent of the uncoded discharges were codable, as compared to 28 percent and 7 percent of the uncoded discharges in Regional Trauma Centers and Specialty Referral Trauma Centers, respectively.

Discussion

This study demonstrates that the current structure of many statewide hospital discharge data systems restricts the use of E-codes for specific subgroups of patients. In Maryland, reporting of E-codes is not required and is done inconsistently across hospitals and patients. In many hospital discharge data systems, including HCFA's UB-82, E-codes can only be included in the discharge abstract as one of five discharge diagnoses.[13] Since ICD E-codes are not used as a basis for reimbursement, they receive low priority in the selection process of the five ICD-9CM codes reported.[14] Thus E-coding rates are lowest among selected groups of patients that are typically characterized by large number of discharge diagnoses, i.e. severely injured patients having multiple injury-related diagnoses, the elderly patients who are likely to have chronic diseases existing prior to the trauma and are more prone to complications leading to additional discharge diagnoses, and the patients with long hospital stays either due to severe injuries or complications. Injuries, preexisting chronic conditions and complications all take precedence

TABLE 1—E-Coding Status by Body-Region of Principal Diagnosis and by ISS, Maryland Trauma Discharges, 1988

	%E-Coded		% No E-Code Codable		% No E-Code Uncodable		Total	
ISS	Head/ Neck	Extremities	Head/ Neck	Extremities	Head/ Neck	Extremities	Head/ Neck	Extremities
1–8	65	54	20	38	15	8	100	100
9–12	61	48	12	25	27	27	100	100
13–15	52	44	3	8	45	48	100	100
16–19	53	24	13	4	34	72	100	100
20–24	26	22	2	5	72	73	100	100
≥25	31	12	3	2	66	86	100	100

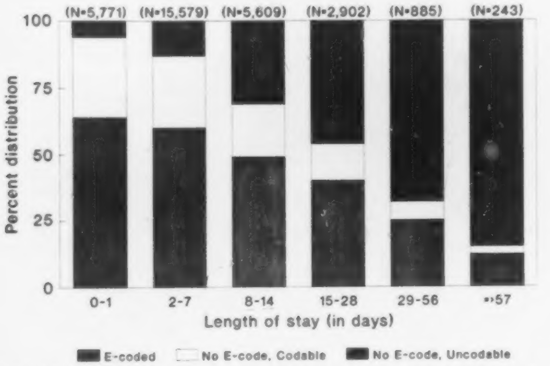

FIGURE 4—E-Coding Status by Length of Hospital Stay, Maryland Trauma Discharges, 1988

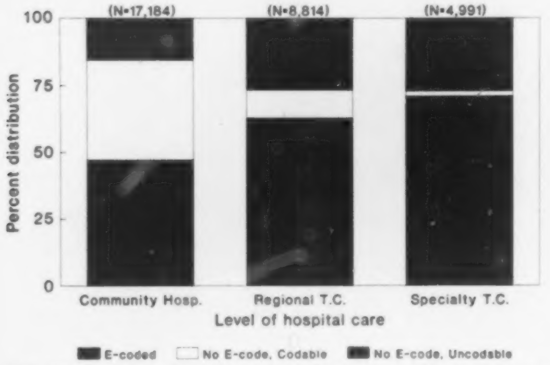

FIGURE 5—E-Coding Status by Level of Hospital Care, Maryland Trauma Discharges, 1988

over cause in coding since they may influence the rate at which hospitals are reimbursed.[14]

In Maryland, the proportion of E-coded trauma discharges has increased from 40 percent in 1979 to 55 percent in 1988. This is likely due to increasing awareness and interest in injury control and Emergency Medical Services (EMS) in the state. Of the uncoded discharges, however, the proportion that were codable has decreased from 98 percent in 1979 to 53 percent in 1988. This trend was most marked in the selected groups of patients described above. This finding suggests that while there is an increasing awareness of the importance of E-coding in the state, there is less potential for including an E-code in the hospital discharge data. This is likely due to the increased attention paid to characterizing the full extent and nature of the injury, complications, and comorbidities and utilization of all five ICD-9CM coding spaces.

Simply increasing the number of fields for recording diagnoses, however, is not an adequate solution. In California, for instance, where a maximum of 25 hospital discharge diagnoses can be recorded,[18] only 40 percent of the trauma discharges in 1983 have been E-coded (MacKenzie EJ, unpublished data). Rather, it will be necessary to mandate E-coding and provide data fields separate from those available for recording diagnoses, preexisting medical conditions, and complications. Mandatory E-coding legislation or regulation, as initiated in New York, California, Wisconsin, and Washington State,[13] and more recently in Rhode-Island and Vermont, provide the means to ensure better compliance with E-coding practice. As has been demonstrated in this study, however, it is essential that separate fields for recording of E-codes be created otherwise it may not be recorded on computerized data bases for those injury cases with multiple diagnoses. There is a need for two E-coding fields in order to adequately characterize the external cause and intent of the injury.[19] Monitoring trends in E-coding rates in these states will be important to assess the extent of compliance and accuracy. Mandating that trauma discharges be E-coded will only be effective if hospital coders are properly trained.[13] Computer software recently developed by the Centers for Disease Control should assist in simplifying E-coding and increase in reliability.[20] In addition, training manuals and courses for E-coding are now available.[19]

Until such time as E-codes are uniformly recorded for all trauma discharges, caution must be exercised when extrapolating results by cause of injury based on only those discharges that are E-coded. This study shows systematic underreporting of external causes of injury in specific trauma patients groups such as the elderly, the severely injured, etc. At the very least, extrapolations should only be made within strata of the population defined by age of the patient and the nature and severity of the injuries sustained. It is also important to recognize that patterns of E-coding may vary over time, by geographic region and by hospital type and coding practices. Gathering cause of injury data from different databases requires attention to the scope and direction of E-code reporting bias that each system may introduce.

Hospital discharge data represent an important and usually the only source of population-based data on serious, nonfatal injuries. Systematic, uniform, and reliable reporting of E-codes in these databases would provide essential information for monitoring trends and patterns of injury, defining subgroups of the population at high risk of injury, and in developing and evaluating injury prevention programs.

ACKNOWLEDGMENTS

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REFERENCES

1. Graitcer PL: The development of state and local injury surveillance systems. J Safety Res 1987; 18:191–198.
2. Fife D, Faich G, Hollinshead W, Boynton W: Incidence and outcome of hospital-treated head injury in Rhode Island. Am J Public Health 1986; 76:773–778.
3. Payne SR, Waller JA: Trauma registry and trauma center biases on injury research. J Trauma 1989; 29:424–429.
4. Lloyd LE, Graitcer PL: The potential for using a trauma registry for injury surveillance and prevention. Am J Prev Med 1989; 5:34–37.
5. McLoughlin E, Langley JD, Laing RM: Prevention of children's burns Legislation and fabric flammability. NZ Med J 1986; 99:804–807.
6. Philipp R: Monitoring accidents and seat belt legislation: HAA data and the use of E-codes in England and Wales. Community Med 1983; 5:235.
7. Runyan CW, Kotch JB, Margolis LH, Buescher PA: Childhood injuries in North Carolina: A statewide analysis of hospitalizations and deaths. Am J Public Health 1985; 75:1429–1432.
8. MacKenzie EJ, Steinwachs DM, Edelstein SL: Utility of uniform hospital discharge data in trauma research and system evaluation. Proc Assoc Adv Auto Med 1988; 32:89–110.
9. ICDMAP—Determining Injury-Severity for Hospital Discharges; A Program to Map ICD-9CM Diagnoses into AIS and ISS Severity Scores. Baltimore: Health Services Research and Development Center, Johns Hopkins University, 1988.
10. MacKenzie EJ, Steinwachs DM, Shankar B: Classifying trauma severity based on hospital discharge diagnoses; Validation of an ICD-9CM to AIS-85 conversion table. Med Care 1989; 27:412–422.
11. MacKenzie EJ, Edelstein SL, Flynn JP: Trends in hospitalized discharge rates for head injury in Maryland, 1979–1986. Am J Public Health 1990; 80:217–219.
12. Committee on Trauma Research: Injury in America. Washington, DC: National Academy Press. 1985
13. Sniezek JE, Finklea JF, Graitcer PL: Injury coding and hospital discharge data. JAMA 1989; 262:2270–2272.
14. Injury Prevention: Meeting the Challenge. The National Committee for Injury Prevention and Control (US). New York: Oxford University Press, 1989.
15. Rice DP, MacKenzie EJ, and associates: Cost of Injury in the United States: A Report to Congress. San Francisco, CA: Institute for Health & Aging, University of California, and Injury Prevention Center, Johns Hopkins University, 1989.
16. Baker SP, O'Neill B, Haddon W, Long WB: The Injury Severity Score: A method for describing patients with multiple injuries and evaluating emergency care. J Trauma 1974; 14:187–196.
17. Annotated International Classification of Diseases, 9th Revision, Clinical Modification. Ann Arbor, MI: Commission on Professional and Hospital Activities, 1986.
18. Office of Statewide Health Planning and Development: Discharge Data. Sacramento, California: California Health and Welfare Agency.
19. Berenholz G: Introduction to ICD-9CM; Injuries and External Causes. Lexington, MA: Berenholz Consulting Associates, October 1987.
20. McClain PW, Pollock DA: A microcomputer program for coding external cause of injury. J Trauma 1989; 29:55–58.

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Benzodiazepine Tranquilizers and the Risk of Accidental Injury

Gerry Oster, PhD, Daniel M. Huse, MA, Shelley F. Adams, MPA,
Joseph Imbimbo, BA, and Mason W. Russell, MAPE

Abstract: To determine whether benzodiazepine tranquilizers increase the risk of accidental injury requiring medical attention, we used pharmacy claims submitted to a large third-party payer to identify 4,554 persons who had been prescribed these agents and a matched control group of 13,662 persons who had been prescribed drugs other than benzodiazepines. We then used diagnoses recorded on claims submitted by medical care providers to identify all accident-related care received by these persons during three months before their first-observed prescription for a benzodiazepine or nonbenzodiazepine agent, respectively, and six months subsequently. We found accident-related care was more likely among persons who had been prescribed benzodiazepines; among these persons, the probability of an accident-related medical encounter was higher during months in which a prescription for a benzodiazepine had recently been filled compared to other months; and persons who had filled three or more prescriptions for these agents in the six months following initiation of therapy had a significantly higher risk of an accident-related medical event than those who had filled only one such prescription. Approximately two-fold risks of accident-related care were found, after controlling for age, sex, and prior utilization. (*Am J Public Health* 1990; 80:1467–1470.)

Introduction

Benzodiazepine tranquilizers were introduced in the United States in 1962 and rapidly supplanted barbiturates as anxiolytic agents of first choice. While they are among the most widely prescribed classes of drugs, there is mounting evidence that their use impairs cognitive performance and psychomotor skills. Persons given benzodiazepines have been found to perform poorly in driving simulator tests relative to those receiving placebo.[1,2] In addition, several forensic studies[3–6] have found a greater than expected number of benzodiazepine users among persons fatally injured in motor vehicle accidents.

Three epidemiologic investigations of impairment related to the use of minor tranquilizers have been reported to date. A study conducted in England over a two-year period found that patients receiving benzodiazepines and other minor tranquilizers were nearly five times more likely to experience a serious motor vehicle accident than those who had not used these drugs.[7] Another study examined persons hospitalized for injuries resulting from a motor vehicle accident and found that persons identified as drivers who were at fault in their accidents were somewhat more likely to have filled a prescription for hypnotics or tranquilizers during the three-month period prior to the accident than those identified as passengers.[8]

The third epidemiologic investigation, a retrospective cohort study using claims data from a large health insurer,[9] compared accident-related medical care among persons who were prescribed benzodiazepines with that of a matched control group who had not been prescribed these agents. Persons prescribed benzodiazepines were more likely to have accident-related care, but also were reported to experience a greater number of nonaccident-related medical encounters, suggesting differences in care-seeking behavior rather than risk of accidental injury. Without data on medical utilization prior to therapy, the effect of differences in care-seeking behavior could not be determined.

To circumvent this difficulty, in the present study we employ a pretest-posttest design[10] to examine three key questions: 1) Are persons who are prescribed benzodiazepine tranquilizers more likely to require accident-related medical care? 2) Is there evidence of a temporal relation between prescriptions for these agents and the likelihood of an accident-related medical encounter? 3) Is there evidence of a relation between the number of such prescriptions and the risk of these encounters?

Methods

Data Source

Data for this study were obtained from the health care claims processing system of Master Health Plus™, a health insurance plan operated by Blue Cross and Blue Shield of Massachusetts with a membership in excess of one million persons. Plan members receive first-dollar coverage for physician services, inpatient and outpatient care, and prescription drugs, with a \$5 copayment for provider services, a \$4 copayment for prescription drugs (\$3 for generics), and a \$500 annual limit on reimbursement for mental health services. All claims for care are provider-submitted, and nondrug claims contain detailed diagnostic information in the form of physician-recorded ICD-9-CM codes. A complete record of medical utilization may be compiled for each plan member during his or her period of enrollment.

To be eligible for inclusion in the present study, members had to be enrolled continuously in the plan during the period April 1986–September 1987. Persons over 65 years of age or otherwise eligible for Medicare benefits were excluded from the study because Medicare claims were not available.

For our sample of persons who were prescribed benzodiazepines (benzodiazepine "users"), we selected all eligible plan members who had at least one pharmacy claim for a benzodiazepine tranquilizer (alprazolam, chlordiazepoxide, clorazepate, diazepam, lorazepam, or oxazepam, either singly or in combination with any other agent) during July 1986–March 1987, and had no claims for any of these agents between April 1986–June 1986. We considered the date of the first-observed pharmacy claim for a benzodiazepine agent to be the date of initiation of benzodiazepine therapy. A total of 4,554 benzodiazepine users were identified and included in the study.

From Policy Analysis Inc. (Oster, Huse, Russell); New England Institute for Healthcare Services Research (Adams); and Electronic Data Systems, Inc. (Imbimbo). Address reprint requests to Gerry Oster, PhD, Policy Analysis Inc., 1577 Beacon Street, Brookline, MA 02146. This paper, submitted to the Journal July 11, 1989, was revised and accepted for publication April 17, 1990.

Three persons who were not prescribed benzodiazepines (benzodiazepine "nonusers") were matched randomly to each user. Nonusers were selected from among those eligible patients who had no pharmacy claims for a benzodiazepine tranquilizer between April 1986–March 1987, were not related by blood or marriage to any patient who had a claim for such an agent during April 1986–March 1987, and had at least one claim for a nonbenzodiazepine agent during July 1986–March 1987. We considered the date of the first-observed nonbenzodiazepine drug claim to be the date of therapy initiation for each nonuser, and we matched nonusers to users on the basis of age, sex, and the calendar month during which therapy was initiated.

For each person in the sample, we then compiled all health care claims for three months prior to the date of initiation of therapy, and for six months subsequent to that date. Every nondrug claim was classified as either accident- or nonaccident-related on the basis of the physician-recorded ICD-9-CM diagnosis codes. Claims with any ICD-9-CM diagnosis code between 800-959 or 990-995 were considered to be accident-related; all other claims were considered nonaccident-related. Emergency visits were identified on the basis of accompanying information noting the type of service rendered (i.e., "emergency medical care").

Univariate Analysis

To examine the relation between benzodiazepine use and overall risk of accidental injury, we considered three outcomes: 1) the occurrence of any accident-related *emergency outpatient visit;* 2) the occurrence of any accident-related *hospital admission;* and 3) the occurrence of an accident-related *medical encounter of any type*. The frequencies of these events among benzodiazepine users and nonusers during the six-month period following initiation of therapy were determined.

To examine evidence of a temporal relationship between benzodiazepine use and risk of accidental injury, among users we compared the frequencies of accident-related medical encounters during months of presumed drug exposure to those during months of presumed nonexposure. A user was presumed to have been "exposed" to benzodiazepines during a given month if a pharmacy claim for one of these agents was observed in that month or the previous month.

Finally, to ascertain whether there is evidence of a relation between the number of benzodiazepine prescriptions filled and the risk of accidental injury, we compared rates of accident-related medical events among users according to their numbers of pharmacy claims for benzodiazepine agents during the six months following initiation of therapy. Two groups of users were defined—those who had only one pharmacy claim for a benzodiazepine agent and those who had three or more such claims—and we compared rates of accident-related medical events between them.

Comparisons of rates were expressed as ratios, or relative risks, and 95 percent confidence intervals were calculated by the Taylor-series method.[11]

Multivariate Analysis

To control for factors other than benzodiazepine use, we fitted a Cox proportional hazards model[12] and included as covariates age, sex, and the logarithms of total accident- and nonaccident-related medical care charges during the three months prior to therapy.

We used a similar proportional hazards model to compare the risk of accident-related medical encounters during months in which a user was presumed to have been exposed to benzodiazepines to all other months. Included in this model were a set of additional binary variables indicating, for each month during the six-month period of follow-up, whether a user was presumed to have been exposed to benzodiazepines in that month.

Finally, we used a similar model to compare outcomes between benzodiazepine users who filled three or more prescriptions for these agents and those who filled only one such prescription. Risks of each accident-related event were estimated as a function of the number of benzodiazepine prescriptions filled, age, sex, and the logarithms of total accident- and nonaccident-related medical-care charges during the three months prior to therapy.

Results

Profile of Benzodiazepine Users and Patterns of Benzodiazepine Use

Benzodiazepine users were more likely to be women than men (65.4 percent versus 34.6 percent), and more than one-half (53.5 percent) were less than 45 years of age. Approximately one-half (48.3 percent) of benzodiazepine users filled only a single prescription for one of these agents, 21.3 percent filled two prescriptions, 11.2 percent filled three prescriptions, and 19.2 percent filled four or more prescriptions over the six-month period of follow-up.

During the three-month period prior to the initiation of therapy, benzodiazepine users were twice as likely as nonusers (95 percent CI: .96, 4.44) to have at least one accident-related emergency outpatient visit (2.4 versus 1.2 per 1,000); 1.7 times as likely (95 percent CI: 1.11, 2.61) to have an accident-related hospital admission (7.2 versus 4.2 per 1,000); and 1.5 times as likely (95 percent CI: 1.29, 1.66) to have an accident-related medical encounter of any type (73.6 versus 50.2 per 1,000.

Accident-Related Care among Benzodiazepine Users and Nonusers

During the six-month period following the initiation of therapy, benzodiazepine users were significantly more likely than nonusers to require accident-related care (Table 1). Compared to nonusers, users were more than twice as likely to have had an accident-related emergency outpatient visit, about 1.6 times more likely to have experienced at least one accident-related hospital admission, and approximately 1.2 times more likely to have had at least one accident-related medical encounter of any type.

We examined rates of accident-related encounters among persons who had no accident-related care during the three-month period prior to the initiation of therapy. Unadjusted relative risks of accident-related events among benzodiazepine users were similar to those reported in Table 1.

TABLE 1—Risks of Accident-related Medical Encounters among Persons Who Were Prescribed Benzodiazepine Tranquilizers ("users") Compared to Those Who Were Not ("nonusers")

Accident-Related Medical Encounter	Monthly Rate per 1,000		Unadjusted Relative Risk (95% CI)	Adjusted Relative Risk* (95% CI)
	Users	Nonusers		
Any emergency outpatient care	1.06	.49	2.16 (1.35,3.50)	2.09 (1.27,3.42)
Any hospital admission	1.39	.88	1.58 (1.07,2.34)	1.27 (.84,1.90)
Any medical encounter	23.31	18.84	1.24 (1.14,1.35)	1.15 (1.05,1.26)

*Hazard ratio adjusted for age, sex, and pretreatment medical care utilization.

For emergency outpatient visits, the unadjusted relative risk was 2.18 (95 percent CI: 1.27, 3.77); for hospital admissions, it was 1.77 (95 percent CI: 1.13, 2.78); and for medical encounters of any type, it was 1.21 (95 percent CI: 1.10, 1.33). Controlling for age, sex, and health care utilization in the three months prior to the initiation of therapy did not change the excess risk of accident-related medical encounters among benzodiazepine users relative to nonusers appreciably (Table 1).

Accident-Related Care by Temporal Pattern of Benzodiazepine Use

Outcomes among benzodiazepine users were examined during 11,389 months of presumed drug exposure (i.e., months in which prescriptions for benzodiazepines had recently been filled) and 15,935 months in which they were presumed unexposed. A greater likelihood of accident-related medical encounters was observed during months of exposure (Table 2). Controlling for age, sex, and health care utilization in the three months prior to treatment, adjusted relative risks were somewhat lower than unadjusted ratios but remained excessive.

Accident-Related Care by Amount of Benzodiazepine Use

Benzodiazepine users who filled at least three prescriptions for these agents (n = 1,384) were more likely to have required accident-related care than those who filled only one such prescription (n = 2,200) (Table 3). Compared to users who filled one prescription, those who filled three or more prescriptions were about 2.4 times more likely to have had an accident-related emergency outpatient visit, and roughly 1.3 times more likely to have had an accident-related medical encounter of any type. The relative risk of an accident-related hospital admission was 1.5. Controlling for age, sex, and health care utilization during the three-month period prior to treatment did not change these risks substantially (Table 3).

Discussion

The results of this study suggest that the use of benzodiazepine tranquilizers increases the risk of accidental injury requiring medical attention. Emergency outpatient care, hospital admissions, and medical encounters of all types were more frequent among persons who were prescribed benzodiazepines than those who were not; among persons who were prescribed benzodiazepines, the likelihood of such care was higher during months in which such a prescription had recently been filled than during other months; and persons who filled three or more prescriptions for benzodiazepines over a six-month period had greater numbers of accident-related medical encounters than those who had filled only one such prescription. Increased risks of accident-related care remain even after controlling for the potentially confounding effects of age, sex, and prior differences in utilization.

TABLE 2—Risks of Accident-Related Medical Encounters among Persons Who Were Prescribed Benzodiazepine Tranquilizers during Months in Which Such a Prescription Was Recently Filled ("exposed") Compared to other Months ("unexposed")

Accident-Related Medical Encounter	Monthly Rate per 1,000		Unadjusted Relative Risk (95% CI)	Adjusted Relative Risk* (95% CI)
	Exposed	Unexposed		
Any emergency outpatient care	1.23	.94	1.31 (.63,2.70)	1.13 (.46,2.77)
Any hospital admission	1.84	1.07	1.73 (.91,3.27)	1.42 (.66,3.04)
Any medical encounter	31.87	17.19	1.85 (1.59,2.16)	1.28 (1.04,1.56)

*Hazard ratio adjusted for age, sex, and pretreatment medical care utilization.

TABLE 3—Risks of Accident-related Medical Encounters among Persons Who Filled Three or more Prescriptions for Benzodiazepine Tranquilizers ("scripts") Compared to Those Who Filled one such Prescription

Accident-Related Medical Encounter	Monthly Rate per 1,000		Unadjusted Relative Risk (95% CI)	Adjusted Relative Risk* (95% CI)
	Three or More Scripts	One Script		
Any emergency outpatient care	1.81	.76	2.38 (1.16,6.04)	2.64 (1.15,6.04)
Any hospital admission	1.57	1.06	1.48 (.70,3.13)	1.42 (.67,3.03)
Any medical encounter	27.70	21.52	1.29 (1.10,1.51)	1.30 (1.09,1.55)

*Hazard ratio adjusted for age, sex, and pretreatment medical care utilization.

We note that approximately one-third of the benzodiazepine users in our sample filled three or more prescriptions for these agents over a six-month period, and that these persons were at highest risk. Compared to persons who were not prescribed benzodiazepines, persons who filled three or more such prescriptions were 3.7 times (95 percent CI: 2.05, 6.68) more likely to have had an accident-related emergency outpatient visit (10.8 versus 2.9 per 1,000), 1.8 times (95 percent CI: 0.99, 3.21) more likely to have had an accident-related hospital admission (9.4 versus 5.3 per 1,000), and 1.5 times (95 percent CI: 1.30, 1.67) more likely to have had at least one accident-related medical encounter of any type (166.2 versus 113.0 per 1,000).

Our control group consisted of patients who received prescription drugs other than benzodiazepines, some of which may also affect the central nervous system. Our findings concerning the increase in risk of accidental injury associated with the use of benzodiazepines may therefore be conservative. Since we do not know whether persons who filled prescriptions for benzodiazepines in fact took any medication and, if so, how much they took and for how long, we may have overestimated drug exposure in our user group. Similarly, there may have been some exposure to these agents among our nonusers. While we excluded all persons with pharmacy claims for benzodiazepines from our nonuser group as well as those related by blood or marriage to these persons, we could not exclude those who had received these agents directly from their physician or those who had obtained these drugs illicitly. Our estimates of relative risk may therefore be conservative.

We considered the possibility that benzodiazepine users simply may be more accident prone than nonusers, due to differences in behavior, lifestyle, occupation, and so forth. We feel that this is unlikely, however; such factors could not account for the *temporal* association between benzodiazepine use and accident-related care that we observed among patients receiving these agents.

Since benzodiazepine tranquilizers often are prescribed for acute anxiety, we also considered the possibility that anxiety itself rather than the prescribed medication may cause users of these agents to be more accident-prone. Accordingly, we identified all persons who had claims for mental health services at any time during the 30 days prior to or following the initiation of benzodiazepine or nonbenzodiazepine therapy, but no such claims previously. When included as a covariate in our proportional hazards model, however, this variable was found to be uncorrelated with patients' risk of accident-related care, and was subsequently dropped. We note also that the results of controlled experiments lend credence to our finding that benzodiazepine use per se increases the risk of accident-related care.

We also considered the possibility that benzodiazepine users simply may be more likely than nonusers to seek medical treatment for minor injuries. Benzodiazepine users have been found to have higher rates of medical encounters unrelated to accidents than nonusers,[8] which suggests that users might be prone to seek professional care more often than nonusers. Again, the temporal association between the filling of prescriptions for benzodiazepines and accident-related encounters cannot readily be explained by differences in care-seeking behavior. Also, the excess in accident-related care among persons who received such prescriptions relative to those who did not remained even after we controlled for prior differences in patterns of health care utilization. Finally, while a greater number of outpatient visits might be related to differences in propensity to seek care, an excess of accident-related hospital admissions among benzodiazepine users can less plausibly be attributed to patients' discretion.

The foremost limitation of our study is that we could observe neither benzodiazepine use nor the occurrence of accidental injuries directly. We inferred the former from the presence in patients' records of pharmacy claims for these agents. We inferred the latter from the presence of nondrug claims with physician-recorded ICD-9-CM diagnosis codes indicating injury as the reason for rendering care, and the assumption that such injuries were accidental and not self-inflicted. To the extent that plan members may have received prescription drugs from other sources, we may have incorrectly identified some p[illegible]ons as nonusers who in fact used benzodiazepines. Whi[illegible]s also possible that some study subjects were treate[illegible] injuries without corresponding claims being submitted to the plan, we believe that this is unlikely. Also, there is no apparent reason why such out-of-plan care should be more frequent among persons who were prescribed benzodiazepines than among those who were not, or vice versa.

A more likely problem deriving from the use of claims data is that some of the accident-related care that we observed in the six-month period following initiation of therapy may in fact have been for follow-up treatment of injuries that occurred prior to therapy. The benzodiazepine user group, for example, may have included persons with pre-existing back strain, for which benzodiazepines are sometimes prescribed. Similarly, the benzodiazepine nonuser group, which was selected from among all persons with nonbenzodiazepine drug claims, may have included patients who were prescribed analgesics for control of prior accident-related pain. We note, though, that when we compared rates of accident-related encounters among persons who had no such care during the three-month period prior to therapy, our results were largely unchanged.

There are important limitations inherent in any study that uses claims data. We note, however, that data bases of the type we have employed are being used increasingly to study outcomes of care: health care claims data, for example, recently have been used to monitor the incidence of birth defects among patients receiving the acne medication, isotretinoin,[13] and to estimate the risks of mortality and reoperation following prostatectomy.[14]

Despite possible limitations, we believe that our study has several important implications. Our findings suggest that the use of benzodiazepine tranquilizers may increase the risk of accidental injury. They therefore corroborate those of previous studies. The conditions for which these agents are prescribed, however, need and deserve treatment. Accordingly, patients should be advised of the potential risks associated with the use of these agents. Also, when appropriate, alternative therapies might be considered, particularly for persons who must operate machinery or a motor vehicle, those who would use alcohol, and those who may require an extended period of therapy.

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REFERENCES

1. Smiley A: Experimental studies of driving performance impairment by medications. *In:* O'Hanlon JF (ed): Consensus Conference on Prescription and Nonprescription Medications, Volume 3: Effects on Driving Performance. Princeton Junction, NJ: Communications Media for Education, 1985.
2. Smiley A, Moskowitz H, Ziedman K: Effects of drugs on driving: driving simulator tests of secobarbital, diazepam, marijuana and alcohol. DHHS Pub. No. ADM 85-1386. Washington, DC: Govt Printing Office, 1985.
3. Korte T, Penttila A, Pikkarainen J: The use of drugs in suspected drunken drivers and victims of traffic accidents. J Clin Chem Clin Biochem 1980; 18:721–722.
4. Bo O, Haffner HFW, Langard O, Trumpy JH, Bredsen JE, Lunde PKM: Ethanol and diazepam as causative agents in road traffic accidents: *In:* Israelstam S, Lambert S (eds): Proceedings of the Sixth International Conference on Alcohol, Drugs and Traffic Safety, Toronto, Ontario, 1974.
5. Garriot JC, DiMaio VJD, Zumwalt RE, Petty CS: Incidence of drugs and alcohol in fatally injured motor vehicle drivers. J Forens Sci 1977; 22:383–389.
6. Williams AF, Peat MA, Crouch DJ, Wells JK, Finkle BS: Drugs in fatally injured young male drivers. Public Health Rep 1985; 100:19–25.
7. Skegg DCG, Richards SM, Doll R: Minor tranquilisers and road accidents. Br Med J 1979; I:917–919.
8. Jick H, Hunter JR, Dinan BJ, Madsen S, Stergachis A: Sedating drugs and automobile accidents leading to hospitalization. Am J Public Health 1981; 71:1399–1400.
9. Oster G, Russell MW, Huse DM, Adams SF, Imbimbo J: Accident- and injury-related health care utilization among benzodiazepine users and nonusers. J Clin Psychiatry 1987; 48 (Suppl):17–21.
10. Cook TD, Campbell DT: Quasi-experimentation: Design and Analysis Issues for Field Settings. Boston: Houghton Mifflin Company, 1979.
11. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research: Principles and Quantitative Methods. Belmont, California: Lifetime Learning Publications, 1982.
12. Lee ET: Statistical Methods for Survival Data Analysis. Belmont, California: Lifetime Learning Publications, 1980.
13. Waldholz M: Study of Accutane and birth defects stirs debate on use. Wall Street Journal April 25, 1988; 4.
14. Wennberg JE, Roos N, Sola L, *et al:* Use of claims data systems to evaluate health care outcomes. Mortality and reoperation following prostatectomy. JAMA 1987; 257:933–936.

A Case-Control Study of the Effectiveness of Bicycle Safety Helmets in Preventing Facial Injury

DIANE C. THOMPSON, MS, ROBERT S. THOMPSON, MD, FREDERICK P. RIVARA, MD, MPH, AND MARSHA E. WOLF, PHD

Abstract: In a case-control study we sought to assess the potential effectiveness of helmets in preventing facial injuries. Our study included 212 bicyclists with facial injuries and 319 controls with injuries to other body areas, who were treated in emergency rooms of five Seattle area hospitals over a one-year period. Using regression analyses to control for age, sex, education and income, accident severity, and cycling experience we found no definite effect of helmets on the risk of serious facial injury (odds ratio 0.81; 95 percent confidence interval = 0.45, 1.5), but protection against serious injuries to the upper face (odds ratio 0.27; 95% CI = 0.1, 0.8). No protection was found against serious injuries to the lower face. The independent effect of helmet use on facial injury was difficult to isolate due to the association of head and facial injuries.

Our results suggest that bicycle helmets as presently designed may have some protective effect against serious upper facial injuries. (*Am J Public Health* 1990;80:1471–1474.)

Introduction

Recently we showed that bicycle helmets reduce the risk of head injury by 85 percent.[1] Facial injuries in the population we studied occur at nearly identical rates (43 per 100,000) as head injuries (45 per 100,000) in bicyclists and can be serious. Bicycle crashes were found to account for 7.1 percent of all facial fractures treated in a large Swedish University hospital over a three-year period.[2]

There are no reported studies on the effectiveness of bicycle helmets in altering the risk of facial injury. Several reports suggest facial injuries are decreased in helmeted motorcyclists and moped riders,[3–5] but motorcycle and moped helmets generally have face pieces whereas traditional bicycle helmets have no face pieces.

The present case control study describes facial injuries resulting from bicycle crashes and evaluates the effect of bicycle helmets on the risk of these injuries.

Methods

Data for the present study were collected as part of a case control study of head injuries resulting from bicycle crashes. The methods have been described in detail elsewhere.[1] In brief, the study was conducted by emergency room surveillance at five major Seattle area hospitals: the Central and Eastside Hospitals of Group Health Cooperative (GHC) of Puget Sound, a large staff model health maintenance organization; University Hospital of the University of Washington; Harborview Medical Center, a regional Level I trauma center; and Overlake Hospital, a community hospital caring for a large proportion of the trauma to the east of Seattle. The records of the medical examiner's office were examined for the study period to ascertain any cyclist deaths occurring at the scene of a crash.

Definitions

Cases were defined as bicyclists who had suffered a facial injury in a bicycle crash and who sought treatment at the emergency room of one of the five study hospitals during the one-year study period December 1, 1986 through November 30, 1987. Facial injury was defined as injury to the area of the face beginning with the eyebrows and extending to the chin, mouth, and teeth. Injuries to the forehead and skull were classified as head injuries and those without concurrent facial injuries were excluded from the facial injury case group.

To address possible overascertainment of minor facial injuries among those individuals who seek care for more serious concomitant injuries, such as head injuries, we used only cases with more serious facial injury, defined as lacerations, fractures of the facial bones, and fractures of the teeth.[6,7]

Patients with serious facial injury were divided into two groups: the upper facial region included the midface, nose and eye/orbit area, the region where helmets would be expected to have the greatest protective effects; the lower facial region included the mouth and chin.

The case groups formed by the above definitions contained bicyclists who had injuries to both the head and facial regions and those who had face injury without concomitant head injuries. It is difficult to separate the possible effect of an intervention (helmet) on different injuries particularly when there is close anatomical association (head and face). We felt the most appropriate definition of a facial injury case group would include all crashing cyclists who sustained a facial injury regardless of the presence of injuries to other areas of the body.

The main comparison group (N = 319) was composed of bicyclists who sought care at the same five emergency rooms for bicycle related injuries other than to the face or head. Head injuries were excluded from the control group, since a cause and effect relationship between bicycle safety helmet use and head injury exists.

A population-based control group (N = 558) consisting of GHC member bicyclists who had experienced a crash in the last year was employed in ancillary analyses. Those under 15 years of age in this control group had significantly higher helmet usage rates at the time of the cycle crash (21.1 percent)

Address reprint requests to Diane C. Thompson, MS, Research Coordinator, Harborview Injury Prevention and Research Center, 325 Ninth Avenue, ZXC-10, Seattle, WA 98104. Dr. Rivara is Director of the Harborview Injury Prevention and Research Center, and is also Professor of Pediatrics and Adjunct Professor of Epidemiology at the University of Washington School of Medicine; Dr. Wolf is Epidemiologist with the Center, and Assistant Professor of Epidemiology at the University; Dr. Robert Thompson is Senior Director of Preventive Care Research, Group Health Cooperative of Puget Sound. This paper, submitted to the Journal November 16, 1989, was revised and accepted for publication April 11, 1990.

as compared to the same age group from the emergency room controls (5.9 percent).[1]

Data Collection

As described elsewhere[1] emergency room cases and controls were identified by weekly surveillance of emergency room logs. All cases and controls were mailed an introductory letter and study consent form explaining the purpose of the study. Detailed questionnaires described earlier[1] were sent to each person with telephone follow-up of nonrespondents. Medical data on injuries was obtained by abstracting the emergency room records.

Statistical Analysis

The statistical package SASPC was used to evaluate descriptive information and prepare crude risk estimates. Unconditional logistic regression techniques were used to estimate the relative risk of facial injury associated with helmet use and to control for multiple confounding factors.[8] The Egret software package was used to carry out logistic regression. The Mantel Haenszel procedure was used to compute age stratified odds ratios in instances where cell sizes were small or included zero.[9]

Results

Seven hundred seventy-six people were treated in the five hospital emergency rooms for bicycling injuries during the study period. Two hundred fifty-six individuals had facial injuries and were potential cases; 368 had injuries involving other parts of the body and were potential emergency room controls. An additional 151 emergency room controls with head injuries were excluded from the analyses. Overall response rate to our questionnaire was 85.0 percent (82.8 percent of cases and 86.7 percent of controls) resulting in 212 cases and 319 emergency room controls available for analysis.

The type and location of the injuries are presented in Table 1. Of the 212 individuals with facial injuries, 136 (64.2 percent) had injuries to one area of the face, 53 (25 percent) had injuries to two areas of the face, 18 (8.5 percent) had injuries to three areas, and 2.3 percent had injuries to four or five areas of the face.

There were three deaths in the study, all of whom were adults with severe head injuries in addition to facial injuries. No bicyclists died at the crash scene.

Demographic Characteristics and Helmet Use of Cases and Controls

As shown in Table 2, cases were younger than controls. There were no important differences between case and control groups by sex, education of head of household, or household income. The distribution of helmet use at time of crash differed between cases and controls. Individuals with any type of facial injury were less likely to have been using helmets at the time of the crash than the control group. There was substantially lower helmet usage among the youngest cyclists. Helmet ownership rates followed a pattern similar to usage rates. Approximately 90 percent of the helmets were of the hardshell variety.

TABLE 1—Type and Location of Facial Injuries Experienced (N = 212 individuals)

	Number and Type of Injury		
Location	Abrasion/Contusion	Laceration	Fracture
Eye/Orbit	23	8	4 (orbit)
Nose	20	12	4
Midface	72	18	7 (zygoma)
Mouth	12	20	33 (teeth & maxilla)
Chin	29	52	4 (mandible)

Notes: Injuries to one area/location of the face are not mutually exclusive of injuries to other areas.

Types of injuries at each location are mutually exclusive.

Crash Characteristics of Cases and Control

Table 3 portrays various aspects of crash severity. Cases were more likely to have hit a stationary obstacle than were controls. Damage to the bicycle was somewhat more frequent for cases. Collisions with motor vehicles, self-reported bicycle speed at the time of crash, and the type of surface impacted was similar for both cases and controls. Cycling experience (kilometers ridden per week, data not shown) was significantly greater in control group.

Risk of Facial Injury Adjusted for Covariates

The focus of our study was to examine the association between bicycle safety helmet use and a set of successively more restrictive and biologically plausible outcomes: all facial injuries, serious facial injuries, and serious upper facial injuries.

The following variables were evaluated using logistic regression analyses: age, sex, cycling experience (kilometers per week cycled), severity (cause of crash, damage to cycle), education, income, and hospital. Only age, as a covariate, accounted for marked changes in computed odds ratios; therefore, we chose to use the simplest model adjusting only for the effects of age. There appeared to be no definite association between safety helmet use and all facial injuries (odds ratio 0.69; 95% CI = 0.41, 1.1) or serious facial injuries (odds ratio 0.81; 95% CI = 0.45, 1.5). When cases with only serious upper facial injuries were examined, a protective effect emerged (odds ratio 0.27; 95% CI = 0.1, 0.8).

Independent Effect of Helmet Use

The analysis of the association between bicycle safety helmet use and facial injury is difficult because some of the association demonstrated between helmet use and facial injury risk is due to the additional association of facial injury and head injury.

Ninety-eight (46.2 percent) of the 212 facial injury cases had a concurrent head injury; 48 (37.8 percent) of the 127 cases with serious facial injury (lacerations, fractured bones or teeth) had a concurrent head injury. The serious facial injury group included a total of 44 individuals with injuries to the upper facial region and 99 individuals with injuries to the lower face; 16 of those individuals had injuries to both regions of the face. Concurrent head injury was present in 27 (61.4 percent) individuals with serious upper facial injury, and in 31 (31.3 percent) of those with serious lower facial injuries.

A series of ancillary analyses were undertaken to attempt to isolate the effect of helmets on facial injuries. The age-adjusted odds ratio for all cases with concurrent head and face injuries (N = 98) was 0.4 (95% CI = 0.18, 0.83). However, when only serious lower facial injuries were examined, the age-adjusted odds ratio was 1.0 (95% CI = 0.6, 2.0) for cases with facial injury only (N = 114) 1.1, (95% CI = 0.57, 1.97).

Finally, we performed analyses utilizing the population control group of GHC cyclists experiencing a crash in the prior year to address the question of protective effect in crashing cyclists in general. Protective effects were found for

TABLE 2—Demographic Characteristics and Helmet Use of Cases and Controls

Characteristics	All Facial Injury Cases (N = 212)		Serious Facial Injuries (N = 127)		Serious Upper Facial Injuries (N = 44)		Emergency Room Controls (N = 319)	
	N	%	N	%	N	%	N	%
Sex								
Male	155	(73.1)	89	(70.1)	30	(68.2)	224	(70.2)
Female	57	(26.9)	38	(29.9)	14	(31.8)	95	(29.8)
Age (years)								
2–14	138	(65.1)	81	(63.8)	23	(52.3)	126	(39.5)
15–24	24	(11.3)	14	(11.0)	6	(13.6)	86	(27.0)
≥25	50	(23.6)	32	(25.2)	15	(34.1)	107	(33.5)
Education of Head of Household								
High School or less	36	(17.0)	17	(13.4)	5	(11.4)	39	(12.3)
College	116	(54.7)	72	(56.7)	25	(56.8)	168	(53.0)
Graduate school	50	(23.4)	31	(24.4)	13	(29.6)	91	(28.5)
Refusals & unknown	10	(4.7)	7	(5.5)	1	(2.3)	21	(6.6)
Household Income								
<$15,000	32	(15.1)	15	(11.8)	9	(20.5)	56	(17.6)
$15–$35,000	65	(30.7)	33	(26.0)	10	(22.7)	79	(24.8)
≥$35,000	89	(42.0)	62	(48.8)	20	(45.5)	138	(43.3)
Refusals & unknown	26	(12.3)	17	(13.4)	5	(11.4)	46	(14.4)
Wearing Helmet at Time of Crash	30	(14.2)	21	(16.5)	4	(9.1)	83	(26.0)

TABLE 3—Characteristics Contributing to Crash Severity

	Cases (N = 212)		Serious Facial Injuries (N = 127)		Upper Serious Facial Injuries (N = 44)		ER Controls (N = 319)	
	N	%	N	%	N	%	N	%
Cause of Crash								
Contact with moving motor vehicle	30	(14.2)	15	(11.8)	7	(15.9)	51	(16.0)
Contact with other moving objects^a	16	(7.6)	9	(7.1)	2	(4.6)	40	(12.5)
Contact with stationary obstacle^b	70	(33.0)	45	(35.4)	16	(36.4)	74	(23.2)
Bicycle malfunction^c								
Riding unsafe bicycle	21	(9.9)	12	(9.4)	2	(4.6)	27	(8.5)
Falls and other	75	(35.3)	46	(36.3)	4	(9.1)	127	(39.8)
Nature of Surface Involved								
Paved (concrete, asphalt)	188	(88.7)	111	(87.4)	37	(84.1)	268	(84.0)
Gravel	14	(6.0)	10	(7.9)	3	(6.8)	21	(6.6)
Dirt/grass	10	(4.7)	6	(4.7)	4	(9.1)	30	(9.4)
Self-Reported Speed at Time of Crash								
Slow (<5 MPH)	70	(33.0)	42	(33.1)	16	(36.4)	96	(30.1)
Moderate (5–15 MPH)	73	(34.4)	46	(36.2)	18	(40.9)	123	(38.6)
Fast (>15 MPH)	48	(22.6)	28	(22.1)	10	(22.7)	87	(27.3)
Unknown	21	(9.9)	11	(8.7)	0	(0)		
Damage to Bicycle								
No	99	(46.7)	63	(49.6)	24	(54.6)	176	(55.2)
Yes	113	(53.3)	64	(50.4)	20	(45.6)	143	(44.8)

a) Bicycles, cars, pedestrians, animals
b) Parked cars, bumps, curbs
c) Chain fell off, flat tire, handlebars came off, no seat

serious upper facial injury; the age-adjusted odds ratio was 0.14 (95% CI = 0.04, 0.47). When facial injuries only were used as the case group, the odds ratio was 0.5, (95% CI = 0.3, 0.9).

Discussion

The results of this study are consistent in suggesting a protective effect of bicycle safety helmets on serious injuries of the upper facial region (lacerations and fractures of the midface, nose, eye/orbit). This conclusion has biological plausibility. The precise magnitude of the protective effect cannot be estimated, since it was not possible to completely disentangle protective effects against facial injury from the effects of head injury occurring concurrently in the same individual.

The significant association of head trauma and facial injury requires careful case and control definition. Head-injured patients were excluded from the emergency room control group because the head injury is directly related to the exposure (helmet use).[1] Their inclusion would have resulted in an underestimation of the protective effect of helmets on facial injuries by selecting for unhelmeted controls. This would be analogous to excluding individuals with lung cancer from the control group in a study of the association of smoking with heart disease.

The most representative facial injury case group is composed of all cyclists with facial injuries including those with injuries to the head or other portions of the body. Equal ascertainment of facial injuries among injured cyclists is important. The presence of a concurrent head injury increases the likelihood that an injured individual seeks medical attention and thus becomes a case. Since head injury is so strongly associated with the lack of helmet use, such a bias can result in an overestimation of the protective effect of helmets by potentially increasing the number of unhelmeted cases. Ascertainment bias was minimized by defining a case group with serious facial injury, i.e. injury serious enough to necessitate medical attention independently of the presence of a head injury. This analysis suggests no association between helmet use and the risk of facial injuries.

We attempted in the analysis to determine the independent effect of bicycle safety helmets on facial injuries. Although there was no effect of helmets on facial injury alone (cases with concurrent head injury excluded) when the emergency room controls were used as comparison, use of the same case group with the population-based control group suggested some protective effect of helmet use (odds ratio 0.5; 95% CI = 0.3, 0.9). This is a more conservative analysis since individuals with head injury and thus low chance of helmet use were excluded. If a helmet effect is present, one would expect to show this effect using the population controls as the comparison group. This is a less seriously injured group of cyclists and has a higher rate of helmet use than the emergency room controls. The demonstration of some protective effect with facial injuries only is important, since it indicates effect on facial injury separate from head injury effects.

Current bike helmets do not have a face bar; consequently one would expect little or no protection against injuries to the lower face. The negative results for serious lower facial injury lend credence to the positive protective effect found for serious upper facial injury.

The strong association between helmet use and decreased risk for serious upper facial injuries is likely explained by a combination of factors: the association of upper facial injuries with head injuries, (61.4 percent of those with serious upper facial injuries) and an actual protective effect of helmets for upper facial injury.

A potential limitation of this study is that unmeasured factors not taken into account in our analysis may have placed controls at lesser risk for striking their faces at the time of the crash.

In conclusion, our study suggests that bicycle helmets as presently designed have little or no effect on the overall risk of facial injury, but appear to provide some protection for serious upper facial injuries. The magnitude of this effect is not precisely quantifiable. This study also illustrates the methodologic problems of isolating exposure or intervention effects on one injury type when multiple simultaneous injuries result from a single event. This problem is inherent to injury research, but much less likely in cancer or cardiovascular investigations.

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REFERENCES

1. Thompson RS, Rivara FP, Thompson DC: A case control study of the effectiveness of bicycle safety helmets. N Engl J Med 1989; 320:1361–1367.
2. Lindqvist C, Sorsa S, Hyrkas T, Santavirta S: Maxillofacial fractures sustained in bicycle accidents. J Oral Maxillofac Surg 1986; 15:12–18.
3. Luna GK, Copass MK, Oreskovich MR, Carr[illegible]o CJ: The role of helmets in reducing head injuries from motorcycle accidents: political or medical issue? West J Med 1981; 135:89–92.
4. Bachulis BL, Sangster W, Gorrell GW, Long WB: Patterns of injury in helmeted and nonhelmeted motorcyclists. Am J Surgery 1988; 155:708–711.
5. Schultz RC: Facial Injuries. Chicago: Year Book Medical Publishers, Inc, Chapter 3, 1988.
6. Karlson TA: The incidence of hospital-treated facial injuries from vehicles. JAMA 1982; 22:303–310.
7. Lovsund P, Lovsund-Johannesson E, Edward S: Traumatic injuries in bicycle accidents among children. Swed Dent J 1988; 12:264–265.
8. Breslow NE, Day NE: Statistical methods in cancer research. Vol. 1. The analysis of case-control studies. IARC scientific Pub. No. 32. Lyon, France: International Agency for Research on Cancer, 1980; 190–246.
9. Mantel N, Haenszel W: Statistical aspects of the analysis of data from retrospective studies of disease. JNCI 1959; 22:719–748.

Association of Hepatitis B Surface Antigen and Core Antibody with Acquisition and Manifestations of Human Immunodeficiency Virus Type 1 (HIV-1) Infection

RACHEL E. SOLOMON, MHS, MARK VANRADEN, MA, RICHARD A. KASLOW, MD, MPH, DAVID LYTER, MD, BARBARA VISSCHER, MD, HOMAYON FARZADEGAN, PHD, AND JOHN PHAIR, MD

Abstract: We examined the associations between seropositivity for hepatitis B virus (HBV) with the presence or development of antibodies to human immunodeficiency virus (HIV-1) and with HIV-1 induced T-helper lymphocyte deficiency or acquired immunodeficiency syndrome (AIDS). Serologic data on HBV and HIV-1, cytometric enumeration of CD4$^+$ lymphocytes, clinical events (AIDS by Centers for Disease Control criteria) and hepatitis B vaccination histories were available on 4,498 homosexual participants in the Multicenter AIDS Cohort Study. Men were classified as to previous infection with HBV and prevalent or incident infection with HIV-1. Although there was an association between seropositivity for HBV infection and HIV-1 infection at enrollment (odds ratios anti-HB$_c$ 2.6; HB$_s$Ag 4.2), the relation between HBV seropositivity and subsequent seroconversion to HIV-1 was weaker (odds ratios 1.3 and 1.6). HIV-1 seroconversion was also associated with a history of certain other sexually transmitted diseases, but predisposing sexual practices did not account for the association between HBV and HIV-1 infection. Seropositivity for HBV infection at entry was not related to initially low or more rapid subsequent decline in T-helper lymphocyte counts and was not associated with an increased incidence of AIDS during 2.5 years of follow-up. History of vaccination against HBV did not appear to decrease susceptibility to HIV-1 infection or to subsequent progression of immunodeficiency. We conclude that prior HBV infection is unlikely to be specifically associated with acquisition of HIV-1 infection and is unrelated to more rapid progression of HIV-1-induced immunodeficiency. (*Am J Public Health* 1990; 80:1475–1478.)

Introduction

Approximately 300,000 new cases of hepatitis B virus (HBV) infection occur every year in the United States alone.[1] Before the spread of acquired immunodeficiency syndrome (AIDS) resulted in altered patterns of sexual behavior, an estimated 60 percent of homosexual males demonstrated serologic evidence of previous hepatitis B infection as measured by antibody to hepatitis B core antigen (anti-HB$_c$), and approximately 5 percent were chronic carriers of hepatitis B surface antigen (HB$_s$Ag).[2] Homosexual males were at 10 to 15 times greater risk of prior HBV infection than men in the general population.[2] Consequently, even before human immunodeficiency virus, type 1 (HIV-1) was established as the cause of AIDS there was considerable interest in a possible direct or cofactorial role for HBV in the AIDS epidemic.[3–5]

In most epidemiologic studies, homosexual men have experienced high rates of infection with both HBV and HIV-1.[6–9] Detailed cross-sectional analysis of men who enrolled in the Multicenter AIDS Cohort Study (MACS), a prospective study of HIV-1 infection in homosexual men, demonstrated that the close association between detectable anti-HB$_c$ or HB$_s$Ag and seropositivity for HIV-1 infection was highly significant and independent of sexual practices commonly predisposing to both infections.[10] Observations of this kind, coupled with the recent description of an effect of the hepatitis B virus protein X on HIV-1 replication in vitro,[11,12] have encouraged speculation about concurrent HBV as a codeterminant for infection or disease with HIV-1. We report here an analysis of possible relations of prevalent HBV infection (anti-HB$_c$ or HB$_s$Ag) to the acquisition and to manifestations of HIV-1 infection in homosexual men.

Methods

The MACS enrolled 4,954 homosexual males without AIDS as defined by CDC criteria[13] in four metropolitan US areas[9,10] from April 1984 through April 1985. The study centers are located in Baltimore, Maryland (Johns Hopkins University), Chicago, Illinois (Howard Brown Memorial Clinic/Northwestern University), Los Angeles, California (University of California), and Pittsburgh, Pennsylvania (University of Pittsburgh). At both entry and follow-up visits interviewers administered a standardized questionnaire, through which we obtained a demographic profile, past medical history, drug and medication history, history of hepatitis B vaccination, psychological assessment, and sexual behavior data.

Serum specimens taken from all men at entry were examined for antibodies to HIV-1, for HB$_s$Ag, and for anti-HB$_c$; at each six-month follow-up visit participants were tested for antibodies to HIV-1. Testing for anti-HIV-1 was performed by enzyme-linked immunoassay (EIA) using licensed, commercially available reagents (DuPont and Genetic Systems), and positive results on the EIA were confirmed by immunoblot assays. Standard EIA or radioimmunoassay techniques using commercially available kits (Abbott Laboratories, Inc.) were used to test for prevalent HBV infection (defined as detectable HB$_s$Ag or anti-HB$_c$). The ordinarily small number of men who may have had antibodies to HB$_s$Ag only were thereby classified as HBV-seronegative. Lymphocytes were phenotyped using standard flow cytometric techniques, as previously described for the MACS,[9] at each of the first three visits for all men and at all subsequent visits for HIV-1-seropositive men, as well as for a subset of HIV-1-seronegative men.

From the National Institute of Allergy and Infectious Diseases (Solomon, VanRaden, Kaslow); the University of Pittsburgh Graduate School of Public Health (Lyter); UCLA Department of Epidemiology (Visscher); Department of Epidemiology, Johns Hopkins School of Hygiene and Public Health, (Farzadegan); Howard Brown Memorial Clinic/Northwestern University Medical School, Department of Medicine (Phair). Address reprint requests to Richard A. Kaslow, MD, MPH, Epidemiology and Biometry Branch, DMID, NIAID, Westwood 739, National Institutes of Health, Bethesda, MD 20892. This paper, submitted to the Journal December 18, 1989, was revised and accepted for publication May 21, 1990.

In our analysis of the effects of hepatitis B infection on HIV-1 infection and accompanying immunodeficiency, we excluded 287 men whose initial serologic status (for HBV or HIV-1) or hepatitis B vaccination status was unknown. Of the remaining 4,667 participants, 531 reported having received hepatitis B vaccine prior to entry, and 362 of those vaccinated were known to be serologically negative for HB$_s$Ag and anti-HB$_c$. These men were analyzed separately because of the diminished likelihood of past or current HBV infection.

To examine the possible relation between prior HBV infection and the degree of cellular immunodeficiency, we compared HBV-infected and -uninfected HIV-1-seropositive men for differences in their numbers of CD4$^+$ and CD8$^+$ lymphocytes at entry and differences in the mean changes in those numbers during 2.5 years of follow-up. Slopes of decline in CD4$^+$ cells were calculated by the least squares method, and differences between slopes for the HBV-infected and -uninfected men were compared with a standard t or a Wilcoxon rank sum test. In addition, a life table analysis of the first 2.5 years of follow-up in HIV-1-seropositive MACS participants was performed to examine the effect of prior HBV infection on the incidence of AIDS.

Associations between outcomes and potential risk factors were individually estimated with odds ratios (OR). Life tables and associated log rank tests were calculated by standard computerized statistical packages (SAS Proc Survfit and Proc Survtest). For cross-sectional multivariate analyses, multiple logistic regression techniques were employed using a standard package (BMDPLR), and adjusted ORs were computed from the coefficients derived from the regression model. For prospective analyses, time-dependent Cox proportional hazards models were applied using a standard package (SAS Proc PHGLM).

Results

The 2,826 MACS participants who were unvaccinated against HBV and had serologic evidence of prior or current HBV infection at entry differed from their 1,310 HBV-seronegative counterparts in HIV-1 seroprevalence at entry and in rates of HIV-1 seroconversion during 6 and 24 months of follow-up (Table 1). For further analysis of the relation between hepatitis B and HIV-1 infections present at baseline, multiple logistic regression was used to adjust for a number of known or suspected baseline correlates of anti-HIV-1 (Table 1). The presence of HB$_s$Ag and/or anti-HB$_c$ remained independently correlated with the presence of HIV-1 at entry (adjusted OR: presence of HB$_s$Ag ± anti-HB$_c$, 4.2; presence of anti-HB$_c$ only, 2.6) (Table 1). As previously reported from the MACS,[10] other sexually transmitted infections such as gonorrhea, anogenital herpes, syphilis, cytomegalovirus infection and others, taken singly or in combination, also showed strong relations (ORs = 1.7–4.1) to baseline HIV-1 seropositivity (data not shown).

In the analysis of the relation between baseline HBV seropositivity and subsequent HIV-1 seroconversion among the 2,421 unvaccinated HIV-1-seronegative men, after adjustment for other potentially related variables, the presence of HB$_s$Ag and of anti-HB$_c$ at entry was less strongly but still related to HIV-1 seroconversion in 83 men who developed HIV-1 antibodies during the six-month follow-up and in 181 who developed HIV-1 antibodies during the 30-month follow-up (Table 1). The analyses also suggested independent relations of magnitude similar to those seen in the cross-sectional analyses (OR = 1.4–1.9) between HIV-1 seroconversion and a history of one or more sexually transmitted infections other than HBV.

To determine the possible role of previous HBV infection in the progression of quantitative T-lymphocyte deficiency, we analyzed T-lymphocyte phenotypes in unvaccinated HIV-1-seropositive participants as follows: 1,348 men with six months, 1,131 men with 18 months, and 929 men with 30 months of follow-up data. The presence of HB$_s$Ag or

TABLE 1—Baseline Prevalence and 6-Month and 24-Month Incidence Rates of HIV-1 Infection According to Baseline HBV Serologic Status in 4,136 Homosexual Men

Baseline HIV-1 Serologic Status (N = 4136)

Baseline HBV Status	Number Studied[*]	HIV-1 Seropositive %	Relative Risk[+] of Baseline HIV-1 Seropositivity: Univariate	Relative Risk[+] of Baseline HIV-1 Seropositivity: Multivariate[†]
HB$_s$Ag+, anti-HBc+or−	282	52.1	6.1	4.2 (2.9–5.9)[§]
HB$_s$Ag−, anti-HBc+	2544	48.3	5.3	2.6 (2.1–3.2)
HB$_s$Ag−, anti-HBc−	1310	15.0	1.0	1.0

Follow-up of Initially HIV-1 Seronegative Men (N = 2421)

Baseline HBV Status	Number Studied[*]	HIV-1 Seropositive %[**]		Relative Risk[†*] of HIV-1 Seroconversion			
				Univariate		Multivariate[†]	
		6m	24m	6m	24m	6m	24m
HB$_s$Ag+, anti-HBc+or−	122	4.1	7.6	2.6	1.7	2.2 (0.8–6.5)	1.3 (0.7–2.7)
HB$_s$Ag−, anti-HBc+	1253	4.9	10.4	3.1	2.3	2.2 (1.2–4.1)	1.6 (1.1–2.3)
HB$_s$Ag−, anti-HBc−	1046	1.6	4.7	1.0	1.0	1.0	1.0

[*] Unvaccinated against hepatitis B.

[+] For baseline and 6-month follow-up analyses, relative risk was measured by odds ratio; for 24-month follow-up analyses, relative risk was measured by hazard ratio (see Methods).

[†] Multivariate analysis adjusted for age, race, study center, education, indices of sexual activity (types and frequencies of specific sexual practices, history of other sexually transmitted infections, a serologic test for syphilis and antibodies to cytomegalovirus) and both parenteral and nonparenteral psychoactive drug use.

[§] 95% confidence intervals by Woolf method.

[**] Cumulative proportions by Kaplan-Meier method.

anti-HB_c showed no consistent relation either to mean initial T-helper ($CD4^+$) and T-suppressor ($CD8^+$) cell counts or to any change in counts of these cell types during the follow-up periods. The absence of any effect on mean decline in $CD4^+$ cell counts persisted after adjustment for initial count. During the entire follow-up, among unvaccinated men the mean six-month declines in counts were: all HB_sAg+ = .0391 cells × 10^9/L; HB_sAg-, anti-HB_c+ = .0452 cells × 10^9/L; and HB_sAg-, anti-HB_c- = .0421 cells × 10^9/L (p = NS). Comparable analyses were performed on T-lymphocyte data available for approximately 18 months from HIV-1-seronegative men, and no relations were found.

During the first 2.5 years of follow-up, AIDS developed in 198 of the 1,532 participants who had HIV-1 antibodies at entry and had not received hepatitis B vaccine. The cumulative 2.5 year life table incidence of AIDS in these men was the same whether serologic evidence of HBV infection was present or absent at entry (Figure 1). In particular, neither men who were positive for HB_sAg at baseline nor men who were negative for HB_sAg but positive for anti-HB_c had a higher incidence of AIDS than men who were negative for both HB_sAg and anti-HB_c. The results were unchanged when stratified by number of $CD4^+$ cells at entry.

To assess the possible effects of HBV vaccine on susceptibility to HIV-1 infection, we analyzed HIV-1 seroconversion rates in HIV-1-negative, HB_sAg-negative, anti-HB_c-negative vaccine recipients versus serologically identical non-recipients. Among 1,320 such participants who had completed at least six months of follow-up, men with and without a history of vaccination reported similar high-risk sexual practices and seroconverted at similar rates during the next six and the next 24 months. In the 24-month period, life table rate of HIV-1 seroconversion was not substantially different between vaccinees (n = 274, 6.0%) and nonvaccinees (n = 1,046, 4.7%) (95% confidence interval of difference: −1.9, 4.5). The slightly higher seroconversion rate in vaccinees is consistent with their slightly higher frequencies of such indicators of high risk as the numbers of sexual partners in the recent past and the proportion of men who had had at least one sexually transmitted infection (data not shown).

At entry, HIV-1-seropositive men with a history of HBV vaccination did not demonstrate a low $CD4^+$ cell count more frequently than serologically identical unvaccinated men. During the 2.5 years of follow-up, the vaccinated men showed a slightly more rapid decline in their $CD4^+$ counts. The difference in mean slopes between vaccinated men (−.075 cells × 10^9/L per six months) and unvaccinated men (−.042 cells × 10^9/L per six months) fluctuated with the statistic examined (p = .044 by t test, p = .017 by the nonparametric Wilcoxon test). During the first 2.5 years of follow-up of initially HIV-1 seropositive men, the life table incidence of AIDS was about 15 percent in those who were HBV-seropositive (Figure 1). In the same interval the incidence was about 20 percent in those who were HBV-seronegative with or without a history of HBV vaccination.

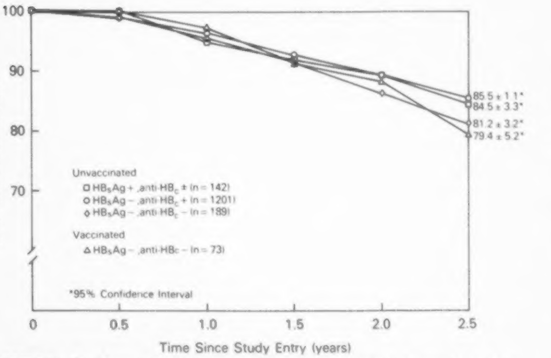

FIGURE 1—Incidence of AIDS among 1605 Initially HIV-1 Seropositive Men, According to Hepatitis B Virus Serologic Status at Baseline

Discussion

Recent evidence that HBV can infect lymphocytes and produce a protein capable of activating HIV-1[11,12] has renewed interest in the early hypothesis that HBV influences susceptibility to or progression of infection with HIV-1.[14,15] Previous studies including our own[10] have demonstrated strong associations between the presence of HIV-1 antibodies at study entry and previous or persistent infection with several sexually transmitted infections, including HBV. Our current investigation confirmed these cross-sectional observations; however, the more powerful longitudinal analysis has provided only meager evidence that previous HBV infection is truly a cofactor for acquisition of infection with HIV-1. We found that HBV infection at baseline was weakly correlated with HIV-1 seroconversion during the next six and the next 24 months. This relation could imply that past or current HBV infection facilitates HIV-1 infection. Since the relation to seroconversion in both time intervals occurred among individuals positive only for anti-HB_c, if there is a true relation, it does not appear to depend closely on the presence of circulating HB_sAg. Although the data could be interpreted as indicative of a causal relation, considering the weakness of the association, we believe that other explanations must be considered equally plausible. One alternative explanation would be that HBV is but one of several sexually transmitted infections nonspecifically capable of facilitating establishment of HIV-1 infection, for example by "priming" T-lymphocytes for penetration or viral replication. Our analyses demonstrated independent relations of similar magnitude between several other sexually transmitted diseases and HIV-1 seroconversion. However, such multiple interrelations could be taken as evidence against any causal contribution of HBV.

To allow for possible temporal variations in the appearance of HIV-1 antibody, we further adjusted for the timing of HIV seroconversion by excluding men who seroconverted early in the follow-up period (within nine months of the baseline evaluation). This did not alter the association between anti-HB_c seropositivity and HIV-1 seroconversion.

Although HBV may persist among persons expressing anti-HB_c in the absence of HB_sAg, a causal relation might have been more readily demonstrable among HB_sAg-positive men. There was no clear evidence of such a causal relation to HIV-1 seroconversion. The absence of this relation was not likely due to the small number of men in this group: the power to detect a difference of two-fold or greater between them and all HB_sAg-negative men was 80 percent.

Our observation that the HIV-1 seroconversion rates were comparable in vaccinated and in unvaccinated seronegative men argues against a protective effect of vaccine and against such a causal role for HBV. A few participants may have erroneously reported HBV vaccination or overestimated its completeness, but it is highly unlikely that misclassified vaccination experience explains these findings.

Our investigation provided no evidence that infection with HBV hastens the appearance of the major immunologic or clinical manifestations of HIV-1 infection. We found no firm associations between positive HBV serology and either altered T-cell phenotype distribution at the time HIV-1 seropositive men entered the study or declining numbers of T-helper cells during the next 24 months. Somewhat divergent results have been reported from the few other studies of the effects of HBV infection on T-cells[16–19] or on clinically significant immunodeficiency[20] in HIV-1-seropositive men. The divergence of findings could be due to the small size of the other studies or to differences in clinical settings, types of patients (i.e. heterosexual vs homosexual) or relative stages of HIV-1 infection. The apparent protection from progressive immunodeficiency in HIV-1-seropositive homosexual male HB$_s$Ag carries in San Francisco[20] was of borderline significance, and the analysis used in that study may not have fully accounted for age, the timing of HIV-1 infection or other factors among the HB$_s$Ag carriers. Our investigation also failed to demonstrate any association, before or after stratification by initial CD4$^+$ cell count, between HBV serologic status and subsequent development of AIDS.

A history of HBV vaccination was not related to altered T-helper lymphocyte counts at entry and probably not related to the slightly more rapid decline in counts subsequently. Our findings, like those of Stevens, *et al*,[20] and others, also suggested that hepatitis B vaccination neither enhances nor retards the development of AIDS. However, our ability to detect an effect of vaccination was limited by the number of vaccinees available for study.

In summary, this prospective investigation of a large number of homosexual men has provided insufficient epidemiologic evidence for and some evidence against the idea that prior or persistent HBV infection facilitates the acquisition of infection with HIV-1, no indication that past or concurrent HBV infection alters progression of HIV-1-induced immunodeficiency, and no suggestion that HBV vaccination in any way protects against either acquisition or progression of HIV-1 infection.

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REFERENCES

1. Centers for Disease Control: MMWR 1987; 36:353–366.
2. Szmuness W, Stevens CE, Harley EJ, Zang EA, Oleszko WR, William DC, Sadovsky R, Morrison JM, Kellnar A: Hepatitis B vaccine: Demonstration of efficacy in a controlled clinical trial in a high-risk population in the United States. N Engl J Med 1980; 303:833–841.
3. Ravenholt RT: Role of hepatitis B virus in acquired immune deficiency syndrome. Lancet 1983; 2:885–886.
4. McDonald MI, Hamilton JD, Durack DT: Hepatitis B surface antigen could harbor the infectious agent of AIDS. Lancet 1983; 2:882–884.
5. Wright T, Friedman S, Altman D: Hepatitis B virus implicated in the pathogenesis of Kaposi's sarcoma in homosexual men. Gastroenterology 1983; 84:1402.
6. Jeffries E, Willoughby B, Boyko WJ, *et al*: The Vancouver Lymphadenopathy-AIDS Study: 2. Seroepidemiology of HTLV-III antibody. Canad Med Assoc J 1985; 132:1373–1377.
7. Mayer KH, Ayotte D, Groopman JE, Stoddard AM, Sarngadharan M, Gallo R: Association of human T lymphotropic virus type III antibodies with sexual and other behaviors in a cohort of homosexual men from Boston with and without generalized lymphadenopathy. Am J Med 1986; 80:357–363.
8. Quinn TC, Piot P, McCormick JB, *et al*: Serologic and immunologic studies in patients with AIDS in North America and Africa. JAMA 1987; 257:2617–2621.
9. Kaslow RA, Ostrow DG, for the Multicenter AIDS Cohort Study Group: The Multicenter AIDS Cohort Study (MACS): I. Rationale, organization and selected characteristics of the participants. Am J Epidemiol 1987; 126:310–318.
10. Chmiel JS, Detels R, Kaslow RA, *et al*: Factors associated with prevalent human immunodeficiency virus (HIV) infection in the Multicenter AIDS Cohort Study (MACS). Am J Epidemiol 1987; 126:568–577.
11. Seto E, Yen TSB, Peterlin BM, Ou J-H: Trans-activation of the human immunodeficiency virus long terminal repeat by the hepatitis B virus X protein. Proc Natl Acad Sci 1988; 85:8286–8290.
12. Twu J-S, Chu K, Robinson WS: Hepatitis B virus X gene activates KB-like enhancer sequences in the long terminal repeat of human immunodeficiency virus 1. Proc Natl Acad Sci 1989; 86:5168–5172.
13. Centers for Disease Control: Revision of the CDC surveillance case definition for acquired immunodeficiency syndrome. MMWR 1987; 36:1S–15S.
14. Laure F, Zagury D, Saimot AG, Gallo RC, Hahn BH, Brechot C: Hepatitis B virus DNA sequences in lymphoid cells from patients with AIDS and AIDS-related complex. Science 1985; 229:561–563.
15. Hirsch MS, Schooley RT, Ho DD, Kaplan JC: Possible viral interactions in the acquired immunodeficiency syndrome (AIDS). Rev Infect Dis 1984; 6:726–731.
16. Hollinger FB, Noonan C, Mansel P, Yoffe B: Potential role of hepatitis B virus (HBV) infection as a cofactor in the development of AIDS. Abstracts of the International Conference on AIDS, Paris, France 1986; 113.
17. Dienstag JL: Immunologic mechanisms in chronic viral hepatitis. *In*: Vyas GN (ed): Viral Hepatitis and Liver Disease. Grune and Stratton, Orlando Fla. 1984; 135–166.
18. Thomas HC, Montano L, Goodall A, DeKoning R, Oladapo J, Wiedman KH: Immunological mechanisms in chronic hepatitis B virus infection. Hepatology 1982; 2:116S–121S.
19. Barnaba V, Musca A, Cordova C, Levrero M, Ruocco G, Albertini-Petroni V, Balsano F: Relationship between T-cell subsets and suppressor cell activity in chronic hepatitis B virus (HBV) infection. Clin Exp Immunol 1983; 53:281–288.
20. Osmond D, Chaisson R, Beasley P, Bachetti P, Moss A: Hepatitis B virus coinfection in homosexual men seropositive for human immunodeficiency virus antibody. III International Conference on AIDS, Washington, DC, June 1–5, 1987, Fl. 5.
21. Stevens CE, Taylor PE, Rubinstein P, Ting RCY, Bodner AJ, Sarngadharan MG, Gallo RC: Safety of the hepatitis B vaccine. N Engl J Med 1985; 312:375–376.

Death in Prison: Changing Mortality Patterns among Male Prisoners in Maryland, 1979–87

MARCEL E. SALIVE, MD, MPH, GORDON S. SMITH, MB, CHB, MPH, AND T. FORDHAM BREWER, MD, MPH

Abstract: The leading causes of death (rate per 100,000 prisoner-years) in Maryland state prisons for the period 1979–87 were circulatory system disease (59), suicide (40), and homicide and legal intervention (30). Acquired immunodeficiency syndrome (AIDS) became the leading cause of death in 1987. Homicides declined after 1980; drug overdose deaths peaked in 1981 and later disappeared. Male inmates have 39 percent lower all-cause death rates than the general population of Maryland after adjustment for age and race. (*Am J Public Health* 1990; 80:1479–1480.)

Introduction

Growing concern with the quality of prison health care has led several groups, notably the American Public Health Association, to develop standards of care for the incarcerated.[1] Despite the recognition that outcomes, including mortality, are useful indicators of quality of care in hospitals[2] and potentially prisons, prison standards do not require routine monitoring. Of over 500,000 inmates in state and federal prisons, 915 males died in the year ending June 1987.[3] National criminal justice statistics[3,4] merely group by type of death, and do not compute rates, make comparisons, or follow trends. Earlier studies[5,6] suggest that inmates have lower mortality than the general population. Descriptive studies have been reported from jails[5,7–10] but not from state prisons where inmates have longer sentences.

This study was designed to: investigate deaths among inmates in the Maryland prison system, examine trends, and compare inmate mortality with that of the general population.

Methods

All male deaths of Maryland state prisoners between January 1, 1979 and December 31, 1987 were studied. Maryland prisons house those persons convicted and sentenced to at least one year; the average stay is 40 months.[11,12] Females comprise only 4 percent of inmates, and were excluded. The prison management information system provided prisoner characteristics. Causes of death obtained from death certificates were classified by nosologists according to the International Classification of Diseases (ICD), 9th edition.[13] Deaths due to illicit drug overdose had been inconsistently classified; for the study they were grouped as unintentional poisoning (ICD E850–E858).

Annual and cause-specific death rates were calculated using prison census figures.[11,12] Indirect standardization used age- and race-specific death rates from Maryland[12,14–16] to calculate expected deaths and standardized mortality ratios (SMR). The 95 percent confidence interval (CI) of the SMR was computed.[17]

Results

Between 1979 and 1987, 204 male inmates died during 93,445 prisoner-years (crude mortality rate 218 per 100,000 prisoner-years). Of the deaths, 140 were from natural causes, 37 suicides, 16 illicit drug overdoses, 9 other unintentional injuries, and two undetermined (Table 1). Leading causes of death (rate per 100,000 prisoner-years) were circulatory system disease (59), suicide (40), homicide and legal intervention (30), malignant neoplasms (28), and unintentional injuries (25).

All-cause mortality peaked in 1980–81 and has since declined paralleling external causes (Figure 1). The natural-cause death rate remained relatively constant, although individual cause rates varied, especially for circulatory system diseases (data available from author on request). Infectious disease death rates were zero until 1984 when they increased rapidly, largely related to human immunodeficiency virus (HIV) infection, and became the leading cause of death in 1987 (65 per 100,000). Other natural causes of death occurred at steady rates. Illicit drug overdose accounted for a rise in external cause death rate that peaked in 1981 (data available from author on request). Homicide and legal intervention deaths had a slight peak in 1980–81 and decreased thereafter. Only one death was due to motor vehicles.

TABLE 1—Comparison of Mortality among Male Prisoners (1979–87) with That of Men in the Maryland General Population (1983) for Major Causes of Death

	Number of Deaths			
Cause of Death (ICD Code)	Observed	Expected	SMR*	(95% CI)
Infectious and parasitic (001–139)	12	6.1	197	(114, 366)
Malignant neoplasms (140–208)	26	53.9	48	(33, 73)
Endocrine and metabolic (240–279)	1	4.6	22	(1, 121)
Circulatory system (390–459)	55	76.8	72	(54, 93)
Respiratory system (460–519)	9	8.6	105	(48, 199)
Digestive system (520–579)	5	11.4	44	(14, 103)
Genitourinary system (580–629)	3	3.3	90	(19, 264)
External causes (E800–E999)	88	119.4	74	(59, 91)
Unintentional injuries (E800–E949)	23	48.6	47	(23, 87)
Suicide (E950–959)	37	20.1	184	(130, 253)
Homicide and legal intervention (E960–E978)	28	47.5	59	(39, 85)
Other causes of death (290–239, 280–389, 630–799)	5	38.6	13	(4, 30)
All causes	204	334.2	61	(53, 70)

*Standardized mortality ratio

When compared with the Maryland general population,

From the Department of Health Policy and Management Johns Hopkins School of Hygiene and Public Health (Salive, Brewer); the Maryland Division of Correction (Salive, Brewer); and the Injury Prevention Center, JHUSHPH (Smith). Address reprint requests to Marcel E. Salive, MD, Center for Medical Effectiveness Research, Agency for Health Care Policy and Research, 5600 Fishers Lane, Rm 18A-19, Rockville, MD 20857. This paper, submitted to the Journal May 3, 1989, was revised and accepted for publication April 9, 1990.

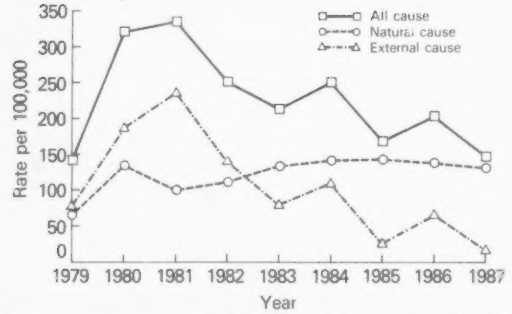

FIGURE 1—All Cause Mortality Rate of Male Inmates over Time, Natural Cause and External Cause Mortality Rates, Maryland State Prison System, 1979–87.

prisoners had lower all-cause mortality for the eight-year period (Table 1) and only suicide and infectious disease deaths were significantly elevated.

Discussion

We examined cause of death in long-term (prison) inmates. Although prisoners had lower all-cause mortality than the general population, significant elevations of suicide and infectious disease deaths were observed. Infectious disease mortality increased due to AIDS, which emerged in 1987 as the leading cause of inmate death. Although fewer than 20 AIDS cases were diagnosed before 1988, the problem will increase since 7 percent of entering male inmates were HIV infected,[18] and HIV transmission has been demonstrated.[19] AIDS prevention and treatment should remain a top correctional health priority. The need for suicide prevention was discussed elsewhere.[12]

Cancer mortality was 46 percent below the comparison population. Circulatory system deaths were also lower, in contrast to prior studies.[6]

External cause mortality overall was 26 percent below that of the general population. Homicide declined steadily and was 41 percent below that of the general Maryland population, although 13 percent of prisoners were convicted murderers. Improved security in the prison, including metal detector installation, is probably responsible for this decline. All injury deaths are potentially preventable and should be investigated.[10]

The epidemic of illicit drug overdose deaths between 1980 and 1982 is probably related to pentazocine (Talwin) use. Six of 16 overdose death certificates reported pentazocine. Pentazocine, although manufactured for oral use, was frequently injected by drug abusers. Its 1983 reformulation added the narcotic antagonist naloxone (inactive when taken orally), reducing overdose deaths nationwide[20,21] and in this prison.

There is a great deal of appropriate concern about poor conditions, riots, murders, outbreaks of illness, and standards of medical care in prisons. Nevertheless, the 39 percent lower Maryland prisoner mortality, similar to that reported in other studies,[5,6] suggests reason for optimism. Decreased mortality may be from a protected environment or lifestyle or a "healthy inmate effect" from selection factors.[5,6] Although data were unavailable from the medical parole program, in our experience premature discharges were rare (none for AIDS) and unlikely to explain lower mortality. Access to medical care may be better in prison than in the general population, which may lead to early detection and treatment of chronic disease. The effect of the availability of correctional officers trained in cardiopulmonary resuscitation needs further study. Limited access to weapons and motor vehicles is likely responsible for low injury mortality, although suicide is elevated. Data limitations preclude an analysis of the development of conditions over time.

The Supreme Court has reiterated the right of prisoners to adequate health care.[22] Any inmate death raises questions about cause, quality of care, and legal responsibility.[5,10] Despite our finding of lower mortality among prisoners, all prisoner death should be investigated to identify remediable health care or safety deficiencies. Improved prison health surveillance is needed for deaths and to determine the effect of prison health services on morbidity.

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REFERENCES

1. Dubler NN (ed): Standards for Health Services in Correctional Institutions, 2nd Ed. Washington, DC: American Public Health Association, 1986.
2. Kahn KL, Brook RH, Draper D, *et al*: Interpreting hospital mortality data: how can we proceed? JAMA 1988; 260:3625–3628.
3. American Correctional Association: Juvenile and adult correctional departments, institutions, agencies and paroling authorities, United States and Canada. College Park, MD: ACA, 1988; xxxii–xxxiv.
4. McGarrell EF, Flanagan TJ (eds): Sourcebook of Criminal Justice Statistics—1984. Pub. No. NCJ-96382. US Department of Justice, Bureau of Justice Statistics. Washington, DC: Govt. Printing Office, 1985.
5. Novick LF, Remmlinger E: A study of 128 deaths in New York City correctional facilities (1971–76). Implications for prisoner health care. Med Care 1978; 16:749–756.
6. Clavel F, Benhamou S, Flamant R: Decreased mortality among male prisoners. Lancet 1987; 2:1012–1014.
7. Smialek JE, Spitz WU: Death behind bars. JAMA 1978; 240:2563–2564.
8. Lanphear BP: Deaths in custody in Shelby County, Tennessee, January 1970-July 1985. Am J Forens Med Pathol 1987; 8:299–301.
9. Copeland AR: Deaths in custody revisited. Am J Forens Med Pathol 1984; 5:121–124.
10. Smith R: Deaths in prison. Br Med J 1984; 288:208–212.
11. Salive ME, Brewer TF: Medical care behind bars: Maryland prison system. Maryland Med J 1989; 38:246–249.
12. Salive ME, Smith GS, Brewer TF: Suicide mortality in the Maryland State prison system, 1979 through 1987. JAMA 1989; 262:365–369.
13. International Classification of Diseases, ninth revision. Clinical modification, DHEW Pub. No. PHS-80–1260. Washington, DC: Govt Printing Office, 1980.
14. National Center for Health Statistics. Vital Statistics of the United States, 1983 Vol. II-Mortality, Pt. A. Hyattsville, MD: Department of Health and Human Services, NCHS, 1987; 244–277.
15. National Center for Health Statistics. Vital Statistics of the United States, 1983 Vol. II-Mortality, Pt. B. Hyattsville, MD: Department of Health and Human Services, NCHS, 1987; 309–314.
16. Bureau of the Census: General Population Characteristics, Final Report 1980, PC80–1–B22, Maryland. Washington, DC: US Department of Commerce, Series P–26, No. 84–52–C, March 1985.
17. Bailar JC, Ederer F: Significance factors for the ratio of a poisson variable to its expectation. Biometrics 1964; 20:639–643.
18. Vlahov D, Brewer F, Munoz A, *et al*: Temporal trends of human immunodeficiency virus type 1 (HIV) infection among inmates entering a statewide prison system, 1985–1987. J AIDS 1989; 2:283–290.
19. Brewer TF, Vlahov D, Taylor E, Hall D, Munoz A, Polk BF: Transmission of human immunodeficiency virus (HIV) within a statewide prison system. JAIDS 1988; 2:363–367.
20. Baum C, Hsu JP, Nelson RC: The impact of the addition of naloxone on the use and abuse of pentazocine. Public Health Rep 1987; 102:426–429.
21. Pokolis A: Decline in abuse of pentazocine/tripelennamine (T's and blues) associated with the addition of naloxone to pentazocine tablets. Drug Alcohol Dependence 1984; 14:135–140.
22. *West v. Atkins*, 108 S. Ct. 2250 (1988).

Long-Term Impact of Smoking Cessation on the Incidence of Coronary Heart Disease

ANNA N. A. TOSTESON, ScD, MILTON C. WEINSTEIN, PhD, LAWRENCE W. WILLIAMS, MS,
AND LEE GOLDMAN, MD, MPH

Abstract: Using a simulation model of the US male population, we estimated the long-term impact that future smoking cessation programs would have on the distribution and occurrence of coronary heart disease in males ages 35–84. For interventions that reduce the number of smokers by 25 percent in 1990, the number of men free of coronary heart disease is projected to increase by 416,787 (0.7 percent) in 2015, and the age-standardized absolute incidence to decline by 2.3 percent. Incidence rates and absolute incidences are projected to fall in men under age 65, but absolute incidence would rise in men over age 65, in large part because of the increased number of men who were at risk for coronary heart disease because of a reduction in non-coronary smoking-related mortality. These trends were more marked for greater smoking reductions and were generally unaffected in a variety of analyses using alternative assumptions, which considered smoking as a risk factor in the elderly, a lag-time before benefits from smoking cessation were realized and secular declines in smoking prevalence. Subject to the assumptions of our model, we conclude that smoking reductions will markedly reduce coronary heart disease, especially in younger age groups, and that this benefit will be slightly offset by a small increase in absolute incidence in elderly men. (*Am J Public Health* 1990; 80:1481–1486.)

Introduction

Smoking is a major risk factor not only for the development of coronary heart disease, but also for other diseases, such as lung cancer and chronic obstructive pulmonary disease.[1,2] Because these risks are to be reduced in persons who quit smoking,[3–6] a growing literature has investigated the effectiveness, economic consequences, and cost-effectiveness of smoking cessation techniques in clinical practice.[7–11]

However, the impact of smoking cessation programs on one outcome, such as coronary heart disease, may be confounded by its impact on the competing risks of developing other smoking-related diseases. We therefore sought to estimate the long-term effect that smoking intervention programs would have on the primary prevention of coronary heart disease in men who are initially free of heart disease at the time of the intervention. Our analysis focused on male smokers, because the risks of developing coronary heart disease as a function of smoking are more precisely measured for males than for females.[12]

Methods

The Coronary Heart Disease Policy Model, which has been described in full detail elsewhere,[13–15] is a state-transition computer-simulation model consisting of three integrated submodels: the Demographic-Epidemiologic Submodel, the Bridge Submodel, and the Disease History Submodel. The Bridge Submodel characterizes subjects for the first 30 days after they develop coronary heart disease, and the Disease History Submodel considers all events that occur among such persons after that 30-day period. Because our analysis focused on the effect of smoking-reduction programs on primary prevention of coronary heart disease, only the Demographic-Epidemiologic Submodel, which simulates the distribution of coronary heart disease risk factors in a population of 35–84 year old persons who are free of coronary heart disease, and computes the annual projected incidence of coronary heart disease on the basis of the simulated distribution, was used.

Each year, persons age 35 years who remain free of coronary heart disease enter the Demographic-Epidemiologic Submodel of the Coronary Heart Disease Policy Model, and persons who turn age 85 exit from the submodel. To exit from the submodel before age 85, persons must either die of non-coronary heart disease causes or develop coronary heart disease (at which time they enter the Bridge Submodel).

The risk factors used by the Demographic-Epidemiologic Submodel to assess each individual's risk of death and of developing coronary heart disease are age, sex, smoking status (no, yes; if yes, average number of cigarettes per day), diastolic blood pressure ($\leq$94 mmHg, 95–104 mmHg, $\geq$105 mmHg), relative weight ($\leq$109% of norm, 110–129% of norm, $\geq$130% of norm), and serum cholesterol ($\leq$249 mg/dl, 250–299 mg/dl, $\geq$300 mg/dl). Based on the categories for each of these factors, the entire United States population that is free of coronary heart disease and is age 35–84 is divided into 5,400 cells, each with a specific value for each factor. For simplicity purposes, however, the model's outputs are collapsed into 10-year age ranges, thus yielding 540 strata.

Assumptions of the Model

The Demographic-Epidemiologic Submodel was initially constructed using the United States population for 1980 by age and sex[16] and the estimated proportion of persons with no history of coronary heart disease.[17] The distributions of smoking status, diastolic blood pressure, relative weight, and serum cholesterol were taken from the Second Health and Nutrition Examination Survey,[18] and the four risk-factor distributions were, for simplicity purposes, assumed to be independent, conditionally upon age range and sex. This assumption may either over- or underestimate the impact that risk factor changes would have on coronary heart disease. For example, a reduction in smoking could cause a decrease in blood pressure or an increase in weight, and an increase in weight could cause an increase in blood pressure.

The number of US residents who enter the model in each subsequent year after 1981 by turning 35 years of age was

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From the Divisions of Clinical Epidemiology, Cardiovascular, and General Medicine, Brigham and Women's Hospital and the Division of Clinical Epidemiology, Beth Israel Hospital and Harvard Medical School; Department of Health Policy and Management, Harvard School of Public Health. Address reprint requests to Lee Goldman, MD, MPH, Division of Clinical Epidemiology, Department of Medicine, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115. This paper, submitted to the Journal December 5, 1989, was revised and accepted for publication May 31, 1990.

estimated from projections of the US Bureau of the Census.[19,20] Risk factor changes with age were estimated from population based cross-sectional data.[18]

For each of the 540 cells, logistic regression coefficients for all-cause mortality and for developing coronary heart disease were based on data from the Framingham Heart Study.[12] The age- and sex-specific coefficients that were used for all risk factors in the Demographic-Epidemiologic submodel were obtained from the Framingham Heart Study's 30-year follow-up, and define the relative odds of death and heart disease for persons with the risk factor in comparison to a baseline group. These raw coefficients were smoothed by age for each sex by means of a weighted least squares regression of the Framingham coefficients on the mid-point of each 10-year age interval, where the weights used were the inverse of the coefficient's standard errors for coronary heart disease incidence. This method gave more weight to the more stable (i.e. smaller standard error) coefficients. The model's coefficients and standard errors that were obtained using this method are compared with the actual Framingham coefficients in Table 1. Note that for persons ≥65 years the smoothed coefficients were negative (−0.0023 for 65–74 years and −0.0136 for 75–84 years) and were therefore set to zero. This approach has been used by others,[8] and is reasonable because several studies indicate non-significant differences in risk of coronary heart disease between smokers and nonsmokers over age 65.[12,21] Overall mortality and coronary heart disease incidence rates were based on US Vital Statistics.[22] Incidence rates for ages 35–74 were assumed to follow those observed in the Framingham Heart Study,[23] with a secular adjustment for the decline in coronary heart disease incidence since the beginning of the Framingham Heart Study,[24] and with extrapolation to ages 75–84 and linear interpolation to smooth the age-specific annual rates.[13]

Intervention

Using the interactive intervention feature of the model, we studied the effect of interventions that produce a substantial reduction in the number of male smokers in the year 1990 by 25%, 50%, and 75%, respectively, on the United States population in the year 2015, 25 years after the reduction was achieved. Interventions were assumed to apply equally across all age groups. The measures that were used to assess the impact of the smoking interventions were the percent change over expected baseline values (i.e., where no smoking intervention is modeled) that the smoking reductions would have on the following measures in the year 2015: a) size of population free of coronary heart disease, b) coronary heart disease incidence rates and c) absolute incidence (the total number of new cases) of coronary heart disease.

TABLE 1—Smoking Coefficients and Standard Errors Used in Demographic-Epidemiologic Model for Determining Risk of Developing Coronary Heart Disease

Age (years)	Model*	Framingham
35–44	0.0316 (0.015)	0.029 (0.010)
45–54	0.0203 (0.010)	0.015 (0.005)
55–64	0.0090 (0.008)	0.016 (0.004)
65–74	0.0 (0.011)	0.002 (0.006)
75–84	0.0 (0.017)	−0.040 (0.023)

*Model coefficients obtained from smoothing Framingham logistic regression function coefficients (see text for details).

Sensitivity Analyses

The variations in our results that would be caused by changes in the coefficients of the logistic risk function for developing coronary heart disease were determined by repeating our analysis four times, using different sets of coefficients each time. The first sensitivity analysis used the actual Framingham coefficients and allowed us to assess the impact that the smoothed coefficients had on our results. The second and third sensitivity analyses used the smoothed coefficients, but first increased and then decreased them by one standard error. These analyses allowed us to assess the effect that interventions on smoking would have if smoking were of more or less importance as a risk factor for coronary heart disease than originally assumed. The fourth analysis examined the impact that a 50 percent smoking reduction would have when smoking was considered as a risk factor for the development of coronary heart disease in the elderly. In this analysis, the smoothed coefficient for persons ages 55–65, 0.009 (Table 1) was applied to persons ≥65.

We also evaluated the impact that delaying the benefits of intervention programs by three years would have on our results. For these analyses, cessation programs begun in 1990 were assumed to have no effect until 1993.

Finally, we evaluated the impact of new smoking interventions on coronary heart disease incidence under the assumption that secular changes in smoking prevalence are already occurring independently of any new intervention programs.[25] For these analyses we estimated the incidence rate and absolute incidence of coronary heart disease in 2015 that would occur if current secular declines continued at a linear rate as they have since 1974[25] through 2000. These estimates were used to compute the incremental change in coronary heart disease incidence that would occur in 2015 if in 1990 a new intervention to reduce smoking by 25, 50, or 75 percent was combined with the projected secular decline through the year 2000.

Results

Increase in Population at-Risk

The projected long-term effect of each smoking intervention within each age group was to increase the number of men at risk for coronary heart disease (i.e. men alive who are free of coronary heart disease) (Table 2). These increases occur in large part because fewer men would die of smoking-related non-coronary diseases if there were reductions in smoking. The increases range from 54,565 to 301,925 persons, 25 years after a 50 percent reduction in smoking prevalence is achieved. Men ages 65–74 in 2015, who were ages 40–49 at the time of the intervention in 1990, had the largest percent increase.

Standardized Absolute Incidence

After adjusting for population differences, the overall reduction in the number of incident coronary heart disease cases in the year 2015, which is 25 years after the smoking reductions are achieved, is projected to range from 2.3% to 5.9% (Table 3). For ages 65–74, there are estimated to be small increases in the number of cases of coronary heart disease under some levels of smoking reduction, and for persons ≥75 years the number of cases is unchanged.

Incidence Rate

The projected baseline rate of coronary heart disease incidence in the United States male population in the year 2015, assuming that cohort-specific risk factor distributions

TABLE 2—Absolute and Percent Change over Baseline in Number of Males at Risk for Coronary Heart Disease (males who are alive and free of coronary heart disease) in the Year 2015 According to Level of Smoking Reduction in 1990

	Level of Smoking Reduction					
Age (years) in 2015	25%	(% Change)	50%	(% Change)	75%	(% Change)
35–44	+28,897	(+0.2)	+54,565	(+0.3)	+77,292	(+0.4)
45–54	+108,846	(+0.6)	+208,283	(+1.1)	+298,800	(+1.6)
55–64	+155,498	(+1.0)	+301,925	(+1.9)	+439,344	(+2.8)
65–74	+91,865	(+1.1)	+180,753	(+2.2)	+266,534	(+3.2)
75–84	+31,681	(+1.0)	+62,881	(+2.0)	+93,567	(+2.9)
Total	+416,787	(+0.7)	+808,407	(+1.3)	+1,175,537	(+1.9)

TABLE 3—Percent Change Over Baseline in Age-Standardized Absolute Incidence of Coronary Heart Disease among Males Free of Coronary Heart Disease According to Level of Smoking Reduction in 1990 (Numbers in Table 2 are obtained by applying incidence rates that occur under each intervention to the baseline population observed in 2015 when no smoking interventions are modeled.)

	Level of Smoking Reduction		
Age (years)	25% (% Change)	50%	75%
35–44	(−9.7)	(−16.7)	(−23.6)
45–54	(−5.2)	(−8.3)	(−13.0)
55–64	(−1.2)	(−2.6)	(−4.0)
65–74	(−0.1)	(+0.4)	(+0.4)
75–84	(0.0)	(0.0)	(0.0)
35–54	(−6.6)	(−10.8)	(−16.2)
35–84	(−2.3)	(−3.9)	(−5.9)

change with age as they have historically, resulted in coronary heart disease incidence rates between 0.29 and 1.96 per 100 (Table 4A). In the year 2015, 25 years after each level of smoking cessation is achieved, there were large decreases in the coronary heart disease incidence rates in the younger age groups. However, a 25 percent reduction in the number of smokers would have no influence on coronary heart disease incidence rates in males over age 65. Larger percent reductions in the number of male smokers would actually *increase* the coronary heart disease incidence rate among 65–74 year olds, while leaving coronary heart disease incidence among 75–84 year olds unchanged. The reasons for these increases are as follows: a) the pool of elderly survivors has been enriched with non-smokers who have slightly less favorable cholesterol levels, blood pressures, and weights; b) many of the at-risk smokers have already developed coronary heart disease; and c) Framingham data indicate that smoking is not a risk factor for coronary heart disease in the elderly (Table 1).

Absolute Incidence

The projected percent change over baseline estimates in the annual absolute incidence of new coronary heart disease cases (i.e. total number) 25 years after each level of smoking cessation is achieved is given in Table 4B. Smoking inter-

TABLE 4—Projected Percent Changes (in parentheses) over Baseline Values for CHD Incidence Rate, Absolute CHD Incidence, and Total CHD Prevalence, 25 Years after each level of Smoking Reduction is Achieved

A. Change in CHD Incidence Rate (per 100 among males free of CHD)

		Change in Incidence Rate for Each Level of Smoking Reduction		
Age (years)	Baseline Incidence Rate	25%	50%	75%
35–44	0.29	(−10)	(−17)	(−24)
45–54	0.64	(−5)	(−8)	(−13)
55–64	1.42	(−1)	(−3)	(−4)
65–74	1.81	(0)	(+1)	(+1)
75–84	1.96	(0)	(0)	(0)
35–54	0.47	(−6)	(−11)	(−16)
35–84	0.96	(−2)	(−4)	(−5)

B. Change in Absolute CHD Incidence (among males free of CHD)

		Change in Absolute Incidence for Each Level of Smoking Reduction		
Age (years)	Absolute Baseline Incidence	25%	50%	75%
35–44	50,471	(−9)	(−16)	(−22)
45–54	119,111	(−4)	(−8)	(−11)
55–64	219,786	(0)	(−1)	(−1)
65–74	149,072	(+1)	(+2)	(+4)
75–84	63,171	(+1)	(+2)	(+3)
35–54	169,582	(−5)	(−10)	(−15)
35–84	601,611	(−1)	(−2)	(−3)

*CHD: Coronary heart disease

ventions did not reduce absolute incidence of coronary heart disease as greatly as they reduced coronary heart disease incidence rates. In particular, the total number of new coronary heart disease cases *increases* for males over age 65. This is because absolute incidence reflects the size of the population at risk, which increases for all age groups as a result of smoking interventions.

Sensitivity Analyses

Changes in Smoking Coefficients—Changes in the smoking coefficients would alter estimates of coronary heart disease incidence rates and absolute incidence equally in younger men. However, under all of our sensitivity analyses (Table 5), the absolute incidence of coronary heart disease would increase in men over age 65. For example, if the actual raw, unadjusted Framingham logistic risk function coefficients were used rather than the smoothed coefficients, the results changed only to a minor degree. The largest difference was in men ages 75–84, in whom the Framingham coefficients are least stable. In this age group, the projected increase in the absolute incidence of coronary heart disease would be even more dramatic (4% higher) with the raw coefficients.

If smoking were not as strong a risk factor for coronary heart disease (i.e. if coefficients were decreased by one standard error), coronary heart disease incidence rates would still decrease for males under age 65, and the effects on absolute coronary heart disease incidence would be approximately one-half as large as under the baseline assumptions. In contrast, if smoking were a more powerful risk factor for coronary heart disease (i.e. if coefficients were increased by one standard error), a 50 percent reduction in the number of smokers in 1990 would result in reductions in coronary heart disease incidence rates by 2015 for *all* age groups. However, under both these scenarios the absolute incidence of coronary heart disease would increase in men over age 65.

When smoking was modeled as a risk factor for development of coronary heart disease in persons ≥65, incidence rates declined in the older age groups. However, absolute incidence still increased (by +1% rather than +2%) in persons ≥65.

Lag Time—Because smoking cessation programs may not produce immediate benefits, the analysis was repeated under the assumption that there is a three-year lag before benefits from smoking cessation would be realized. When the effects of smoking cessation programs begun in 1990 were delayed until 1993, the results changed by less than 1 percent.

Consideration of Future Secular Changes—When the analysis considered the current secular changes in smoking prevalence and assumed that rates of change would continue through the year 2000,[25] the incidence rates and absolute incidences in 2015 were different from those that would pertain if current secular trends do not continue. For example, a continuation of current secular rates of change would be about equivalent to a new intervention that yielded a 25 percent reduction in smoking in 1990.

When the secular decline through 2000 was used as the baseline for estimating the effects of new intervention programs to reduce smoking rates in 1990 by an additional 25, 50, or 75 percent, the effects of the new programs were dampened, but followed the same patterns previously reported. For example, the incremental impact of a 50 percent reduction in the number of smokers in 1990 when combined with a continuation in the secular decline through 2000 was less than the incremental impact of a 50 percent reduction in smoking rates if there are no secular declines, but greater than the incremental impact of a 25 percent reduction in smoking rates if there are no secular declines independent of

TABLE 5—Percent changes[*] over baseline values (i.e., no smoking intervention) for incidence rate and absolute incidence of CHD under alternate logistic risk function coefficients when a 50% decrease in smoking is achieved in 1990.

A. Change in Incidence Rate of CHD in 2015

	Coefficients Used in Logistic Risk Function				
Ages (years)	Smoothed Coefficients %	Unadjusted Coefficients %	Smoothed −1 S.E. %	Smoothed +1 S.E. %	Smoothed Elderly[†] %
35–44	−17	−17	−7	−28	−17
45–54	−8	−6	−5	−14	−8
55–64	−3	−5	−1	−5	−3
65–74	+1	0	0	−2	−2
75–84	0	+5	0	−1	−2

B. Change in Absolute Incidence of CHD in 2015

	Coefficients Used in Logistic Risk Function				
	Smoothed Coefficients %	Unadjusted Coefficients %	Smoothed −1 S.E. %	Smoothed +1 S.E. %	Smoothed Elderly[†] %
35–44	−16	−15	−8	−25	−16
45–54	−8	−5	−4	−12	−8
55–64	−1	−4	+1	−3	−1
65–74	+2	+2	+1	+2	+1
75–84	+2	+6	+1	+3	+1

[*] Note that changes are calculated relative to baseline simulations using the type of coefficients specified. For example, the −28% change in the incidence rate for 35–44 year olds that is observed when smoothed +1 S.E. coefficients are used, was calculated relative to baseline simulations that used the smoothed +1 S.E. coefficients. [†] Smoothed elderly refers to analyses done using smoothed coefficients (Table 1), with coefficients in persons ≥65 of 0.009 (the same coefficient as in the 55–64 age range).

new interventions for ages 35–64. For ages ≥65 years, the incremental impact of a 50 percent reduction when combined with a secular decline was identical to the impact of a 50 percent reduction when no secular declines were modeled.

Discussion

It is important to emphasize that because smoking is a risk factor for the development of many debilitating diseases, including chronic obstructive pulmonary disease and lung cancer, the ability of smoking cessation to improve the overall health of the population is clear. Our study is distinct from other studies, which have sought to measure the effects of risk factor interventions on coronary heart disease mortality and incidence,[3,5,6,21,26–28] because we have examined the long-term, steady-state, population wide impact that smoking cessation can have in the primary prevention of coronary heart disease among US males ages 35–84 who are free of coronary heart disease.

Our results indicate that interventions at a variety of levels would reduce the incidence rate of coronary heart disease for males under age 65, but that incidence rates would increase or remain unchanged for males ≥65 years of age. Note that although our baseline model, which was based on Framingham data, did not consider smoking to be a risk factor for the development of coronary heart disease among males ≥65 years,[12] males in this age group in 2015 were ages 40–59 at the time of the smoking intervention. Thus, they benefited from smoking reductions for at least six years before reaching age 65.

The fact that smoking interventions had the largest impact in reducing coronary heart disease incidence in the younger age groups is consistent with reports in the literature from several populations to indicate that smoking is a stronger relative risk factor for coronary heart disease in the young than in the elderly. The Framingham Heart Study reported incidence rates showing that non-smokers had a higher incidence of coronary heart disease than did smokers over the age of 65, however these differences were not statistically significant.[29] A study of British physicians by Doll and Peto[21] reported similar findings. Hennekens, *et al*,[30] noted that although the relative risks of coronary heart disease associated with smoking may decrease with age, the absolute risk increases. This phenomenon may be explained by the higher background risks of coronary heart disease that are associated with aging, and because many of the "at-risk" smokers may have already died from coronary heart disease or other smoking-related causes.[30,31]

A more recent analysis of data from the Framingham Heart Study indicated that smoking was of borderline significance as a risk factor for coronary heart disease in persons age 65 and older after adjusting for systolic blood pressure, left ventricular hypertrophy, total serum cholesterol, blood glucose, and Metropolitan relative weight.[31] A study of men under age 55 showed decreased risks of myocardial infarction after quitting smoking.[3] A similar phenomenon was reported for elderly men who quit smoking, but the endpoint was coronary heart disease mortality rather than myocardial infarction and this study included men with preexisting coronary heart disease.[5,6] On the basis of these reports and a prospective study that identified smoking as a risk factor for the development of new coronary heart disease in the elderly,[32] we undertook a sensitivity analysis, which modeled smoking as a risk factor in persons ≥65. Although decreases in incidence rates were observed for males ≥65, the intervention still impacted more substantially on the younger age groups.

One striking finding from our analysis is the small *increase* observed in the incidence of coronary heart disease among males ≥65 years of age that occurs under some of the smoking interventions, an effect that was observed even when smoking was modeled as a risk factor in the elderly. These increases emphasize that smoking interventions delay, but do not prevent, the development of coronary heart disease in the male population.

For all smoking interventions, decreases in absolute incidence were less pronounced than were decreases in the incidence rates for males <65 years, because of the larger population at-risk for coronary heart disease as a result of the decrease in smoking-related deaths under each intervention. In addition, there were increases in absolute incidence ranging from 1–4 percent for males ≥65 years under various scenarios. Thus, although decreases in the number of cases of new coronary heart disease would result from smoking interventions for younger persons, there would be a net increase in the number of incident cases of coronary heart disease for males ≥65 years of age.

On the basis of data from the literature, Goldman and Cook[33] estimated that the short-term reduction in coronary heart disease mortality due to an approximate 15 percent decrease in the number of smokers would be between 4.2 and 6 percent. Our results are somewhat different because they represent the differences observed in coronary heart disease incidence and absolute incidence in all men ages 35–84 free of heart disease 25 years after the smoking reduction was achieved. The overall standardized long-term impact of smoking interventions among males aged 35–84 was to reduce the total number of incident coronary heart disease cases by 2.3 percent when smoking rates were reduced 25 percent, by 3.9 percent when smoking rates were reduced by 50 percent, and by 5.9 percent when smoking rates were reduced by 75 percent. These reductions are less than might have been expected.

The Multiple Risk Factor Intervention Trial,[26] which studied men ages 35–57 over a six-year period, estimated that their observed 14 percent difference in smoking rates between the usual care and special intervention groups had the potential to produce a 5 percent difference in the number of coronary heart disease events. Our results for men ages 35–54 at 25 years after a 25 percent reduction in smoking rates was achieved indicate an age-standardized 6.6 percent difference in incident coronary heart disease cases. This estimate reflects the longer-term steady-state projected impact of smoking cessation on coronary heart disease incidence.

Our model depends in part on the accuracy of the National Vital Statistics data on which it is based. Although such data indicate a continued decline in ischemic heart disease (ICD Codes 410–414) mortality since 1980, a good portion of this decline is offset by increases in deaths due to diagnoses such as cardiac arrest and congestive heart failure.[22] These trends suggest that changes in coding practices since the institution of the revision of the ICD criteria in 1975[34] may have had an effect on the decline in deaths that are classified as due to ischemic heart disease.

In addition, the prevalence of ischemic heart disease, as estimated from the updates of the Health Interview Survey,[35] has increased by more than 10 percent since 1980. These trends, in the face of declining coronary risk factors, are consistent with more aggressive, early diagnosis of coronary disease rather than a true increase in atherogenesis. These issues indicate that the Coronary Heart Disease Policy Model will always be addressing a moving target, and thus we plan periodic updates as sufficient epidemiologic data on coronary disease incidence, prevalence, deaths and risk factor distribution become available, usually with a lag time of five or more years.

We did not explicitly consider the economic implications of smoking cessation. Oster, *et al*,[8] have predicted that the increased expenditures attributable to longer life-expectancy under smoking cessation programs are likely to be similar to the savings obtained from reductions in smoking-related illnesses, and Warner[7] has pointed out that the quality of life for non-smokers is likely to be higher than that of smokers. It is therefore not surprising that a variety of smoking interventions have been shown to be cost-effective.[11,36] Warner[7] also stated that although the incidence of cardiovascular disease would decrease in a smoke free society, the age-unadjusted incidence might increase. Our results provide further evidence in support of this conjecture. We conclude that although smoking interventions will not decrease the rate of coronary heart disease incidence for all age groups, the reduction in the number of cases of coronary heart disease among younger persons is substantial, and will offset the slight increases in the older age groups.

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REFERENCES

1. Wilhelmsen L: Coronary heart disease: Epidemiology of smoking and intervention studies of smoking. Am Heart J 1988; 115:242–249.
2. Fielding JE: Smoking: Health effects and control (First of Two Parts). N Engl J Med 1985; 313:491–498.
3. Rosenberg L, Kaufman DW, Helmrich SP, Shapiro S: The risk of myocardial infarction after quitting smoking in men under 55 years of age. N Engl J Med 1985; 313:1511–1514.
4. Ockene JK: The influence of medical care on smoking cessation: Treatment of smoking to prevent coronary heart disease. *In*: Higgins MW, Luepker RV (eds): Trends in Coronary Heart Disease Mortality. The influence of medical care. New York: Oxford University Press 1988; 131–142.
5. Jajich CL, Ostfeld AM, Freeman DH Jr: Smoking and coronary heart disease mortality in the elderly. JAMA 1984; 252:2831–2834.
6. Hermanson B, Omenn GS, Kronmal RA, Gersh BJ, and Participants in the Coronary Artery Surgery Study. Beneficial six-year outcome of smoking cessation in older men and women with coronary artery disease: Results from the CASS registry. N Engl J Med 1988, 319:1365–1369.
7. Warner KE: Health and economic implications of a tobacco-free society. JAMA 1987; 258:2080–2086.
8. Oster G, Colditz GA, Kelly NL: The economic costs of smoking and benefits of quitting. Lexington, MA: Lexington Books, 1984.
9. Kottke TE, Battista RN, DeFriese GH, Brekke ML: Attributes of successful smoking cessation interventions in medical practice: A meta-analysis of 39 controlled trials. JAMA 1988; 259:2882–2889.
10. Eraker SA, Becker MH, Strecher VJ, Kirscht JP: Smoking behavior, cessation techniques, and the health decision model. Am J Med 1985; 78:817–824.
11. Cummings SR, Rubin SM, Oster G: The cost-effectiveness of counseling smokers to quit. JAMA 1989; 261:75–79.
12. US Department of Health and Human Services: The Framingham Study: An epidemiologic investigation of cardiovascular disease. Some risk factors related to the annual incidence of cardiovascular disease and death using pooled repeated biennial measurements: Framingham Heart Study 30-year follow-up. Section 34, NIH Pub. No. 87-2703. Bethesda, MD: National Heart, Lung and Blood Institute, 1987.
13. Weinstein MC, Coxson PG, Williams LW, Pass TM, Stason WB, Goldman L: Forecasting coronary heart disease incidence, mortality, and cost: The Coronary Heart Disease Policy Model. Am J Public Health 1987; 77:1417–1426.
14. Goldman L, Weinstein MC, Williams LW: Relative impact of targeted versus population-wide cholesterol interventions on the incidence of coronary heart disease: Projections of the Coronary Heart Disease Policy Model. Circulation 1989; 80:254–260.
15. Edelson JT, Weinstein MC, Tosteson ANA, Williams L, Lee TH, Goldman L: Long-term efficacy and cost-effectiveness of various initial mono-therapies for mild to moderate hypertension. JAMA 1990; 263:407–413.
16. US Bureau of the Census: Estimates of the civilian population of the United States by age, sex, and race: 1980–83. Current population reports, population estimates and projections, series P-25, number 949. Washington, DC: Govt Printing Office, 1984.
17. National Center for Health Statistics: Unpublished data from the Health Interview Survey, 1979.
18. National Center for Health Statistics: Unpublished data from the Second Health and Nutrition examination Survey, 1976–80.
19. US Bureau of the Census: Preliminary estimates of the population of the United States by age, sex, and race, 1970–1981. Current Population Reports, Population Estimates and Projections, series, P-25, number 922. Washington, DC: Govt Printing Office, 1982.
20. US Bureau of the Census: Projections of the population of the United States by age, sex, and race: 1982–2050. Current Population Reports, Population Estimates and Projections, Series P-25, number 922. Washington, DC: Govt Printing Office, 1982.
21. Doll R, Peto R: Mortality in relation to Smoking: 20 years observation on male British doctors. Br Med J 1976; 2:739–748.
22. National Center for Health Statistics: Vital statistics of the United States Vol II, Part A: Mortality, 1979–1981. Washington, DC: Govt Printing Office, 1981.
23. US Department of Health, Education, and Welfare: The Framingham Study: An epidemiologic investigation of cardiovascular disease. Some characteristics related to the incidence of cardiovascular disease and death, 18-year follow-up. Section 30. Bethesda, MD: Public Health Service, 1973.
24. Pell S, Fayerweather WE: Trends in the incidence of myocardial infarction and in associated mortality and morbidity in a large employed population, 1957–1983. N Engl J Med 1985; 312:1005–1011.
25. Pierce JP, Fiore MC, Novotny TE, Hatziandreu EJ, Davis RM: Trends in cigarette smoking in the United States: Projections to the year 2000. JAMA 1989; 261:61–65.
26. Multiple Risk Factor Intervention Trial Research Group: Coronary heart disease death, nonfatal acute myocardial infarction and other clinical outcomes in the multiple risk factor intervention trial. Am J Cardiol 1986; 58:1–13.
27. The Pooling Project Research Group: Relationship of blood pressure, serum cholesterol, smoking habit, relative weight, and ECG abnormalities to incidence of major coronary events: final report of the Pooling Project. J Chronic Dis 1978; 31:201–306.
28. Blackburn H, Watkins LO, Agras WS, Carleton RA, Falkner B: Task Force 5: Primary prevention of coronary heart disease. Circulation 1987; 76(Suppl I):I-164–I-167.
29. US Department of Health and Human Services: The Framingham Study: An epidemiologic investigation of cardiovascular disease. Some characteristics related to the incidence of cardiovascular disease and death: Framingham Heart Study, 18-year follow-up. Section 30. Bethesda, MD: Public Health Service, 1973.
30. Hennekens CH, Buring JE, Mayrent SL: Smoking and aging in coronary heart disease, *In*: Bosse R, Rose C (eds): Smoking and Aging. Lexington, MA: Lexington Books, 1984.
31. Harris T, Cook EF, Kannel WB, Goldman L: Proportional hazards analysis of risk factors for coronary heart disease in individuals aged 65 or older: The Framingham Heart Study. J Am Geriatr Soc 1988; 36:1023–1028.
32. Aronow WS, Herzig AH, Etienne F, D'Alba P, Ronquillo J: 41-month follow-up of risk factors correlated with new coronary events in 708 elderly patients. J Am Geriatr Soc 1989; 37:501–506.
33. Goldman L, Cook EF: The decline in ischemic heart disease mortality rates: An analysis of the comparative effects of medical interventions and changes in lifestyle. Ann Intern Med 1984; 101:825–836.
34. World Health Organization: Manual of the international statistical classification of diseases, injuries and causes of death. Based on the recommendations of the ninth revision conference, 1975. Geneva: World Health Organization, 1977.
35. National Center for Health Statistics: National Health Interview Survey. Vital and Health Statistics. Series 10, Number 164. Pub. No. PHS87-1592. Hyattsville, MD: Department of Health and Human Services, October 1987.
36. Oster G, Huse DM, Delea TE, Colditz GA: Cost-effectiveness of nicotine gum as an adjunct to physician's advice against cigarette smoking. JAMA 1986; 256:1315–1318.

Overcoming Potential Pitfalls in the Use of Medicare Data for Epidemiologic Research

ELLIOTT S. FISHER, MD, MPH, JOHN A. BARON, MD, MSC, DAVID J. MALENKA, MD,
JANE BARRETT, MSC, AND THOMAS A. BUBOLZ, PHD

Abstract: We used Medicare data bases and US Census data to address two questions critical to the use of Medicare files for epidemiologic research. First, we examined the degree to which the population enrolled in the Medicare program is similar to the elderly resident population of the United States, as estimated by the US Census. We found small differences in the total population estimates but substantial differences by age and race. Second, we found that among Medicare enrollees, physician claims identify a small proportion of hip fracture cases which are not documented in the hospital discharge files. This proportion varies by age, region, and state within the United States. Calculation of rates based on Medicare hospital discharge data, and probably other hospital discharge data sets as well, must take these limitations into account. Use of all available Medicare data files can overcome these limitations. (*Am J Public Health* 1990; 80:1487–1490.)

Introduction

Administrative health care data bases, such as those maintained by the Health Care Financing Administration (HCFA) for the Medicare program, provide an increasingly accessible and widely used source of data for health care research. Medicare files have been used to study disease incidence and risk factors,[1–3] to document regional variations in treatment patterns,[4,5] to study hospital-associated mortality rates and quality of care,[6] and to evaluate the outcomes of specific surgical and medical treatments.[7,8]

Most concern about the validity of research findings based on the Medicare files has focused on issues related to the accuracy of diagnostic coding.[9–11] In this paper we highlight several other potential limitations of the Medicare data relevant to epidemiologic research by addressing two questions. First, can one assume that the population captured in Medicare files is the same as the elderly population of the United States? Second, do the Medicare hospital claims data identify all discharge records for Medicare enrollees who were hospitalized for a specific disease? The answers to these questions highlight how the careful use of Medicare data can overcome these limitations.

Methods

Population Estimates

We used US Census Bureau survey data and Medicare data files to estimate the age, sex, and race-specific counts of United States residents and Medicare enrollees who were over the age of 65 and were alive on July 1, 1985. Census estimates were taken from the Current Population Estimates.[12] Estimates for the Medicare eligible population were determined by using the 5 percent National HISKEW (live) file maintained by the Health Care Financing Administration. This file is a random sample of the Medicare population, based on the terminal two digits of the social security number and records the birth date, state of residence, race, gender, and Medicare eligibility status of beneficiaries who have been eligible for Medicare at some point and are not known to have died. (Deaths are recorded in a different file.) We excluded Medicare eligibles listed as residing outside the 50 States. We then determined age, sex, and race-specific counts of individuals in the HISKEW files generated on 12/31/84 and on 12/31/85. We averaged these counts and multiplied by 20 to estimate the Medicare population alive on July 1, 1985.

Hip Fracture Case Ascertainment

We searched the 5 percent National MEDPAR (hospital discharge) files for all discharges in calendar year 1985 with a discharge diagnosis of hip fracture (ICD-9-CM code 820.0–820.9) and the 5 percent Medicare Part B (physician and other provider) files for all claims specifically documenting the treatment of an acute hip fracture during calendar year 1985 (Current Procedure Terminology codes in the range 27230 to 27248). We developed a detailed algorithm to define acute hip fractures based on all claims submitted during the calendar year for potential cases, relying on both diagnosis and procedure codes and the dates of the related claims.[13] To be included, cases had to have at least one hospital claim specifying the diagnosis, or two Part B claims supporting the diagnosis of hip fracture. We excluded potential cases for whom the only evidence of treatment for the fracture was provided by a single Part B claim (3 percent of candidate cases). This decision was based on extensive review of all claims for a sample of these potential cases. Hip fracture cases were excluded if there was evidence that the treatment was provided for an earlier fracture or that the fracture resulted from metastatic cancer. We applied this algorithm to the data obtained from the 1985 5 percent national sample of Part A and Part B claims.

Results

Population Estimates

We compare the US Census estimate of the elderly population with our estimate of the Medicare population in Table 1. Overall, the Medicare population, as estimated from the HISKEW file, represents approximately 96 percent of the US Census estimate of the elderly population, but the proportion enrolled in Medicare varies sharply by both age and race. Among Whites, only 90 percent of the estimated elderly population between the ages of 65 and 69 is enrolled in Medicare and the proportion enrolled increases with age. Among Blacks, only 79 percent of the estimated population

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From the Department of Medicine, Dartmouth Medical School (Fisher, Baron, Malenka); Department of Community and Family Medicine at Dartmouth (all authors); and the Department of Veterans Affairs Medical Center, White River Junction, Vermont (Fisher). Address reprint requests to Elliott S. Fisher, MD, MPH, Department of Community and Family Medicine, Dartmouth Medical School, Hanover, NH 03756. This paper, submitted to the Journal May 9, 1990, was revised and accepted for publication August 12, 1990.

TABLE 1—Comparison of Population Estimates based on Medicare Enrollment File (HISKEW) and US Current Population Survey (counts in thousands)

	Male				Female				All			
Race/Age	HSK	CNS	H/C	C-H	HSK	CNS	H/C	C-H	HSK	CNS	H/C	C-H
White												
65–69	3425	3821	0.90	396	4196	4615	0.91	419	7620	8436	0.90	816
70–74	2705	2899	0.93	194	3674	3914	0.94	240	6380	6813	0.94	433
75–79	1797	1924	0.93	127	2880	3044	0.95	164	4677	4968	0.94	291
80–84	1021	1051	0.97	30	1998	2001	1.00	3	3019	3052	0.99	33
80+	674	695	0.97	21	1760	1779	0.99	19	2434	2474	0.98	40
Total	9623	10390	0.93	767	14508	15313	0.95	805	24131	25743	0.94	1612
Black												
65–69	287	361	0.79	74	374	472	0.79	98	661	833	0.79	172
70–74	230	258	0.89	28	331	373	0.89	42	561	631	0.89	70
75–79	152	172	0.89	20	250	270	0.93	20	403	442	0.91	39
80–84	87	84	1.04	−3	164	149	1.10	−15	251	233	1.08	−18
85+	65	65	0.99	0	146	140	1.04	−6	210	205	1.03	5
Total	821	940	0.87	119	1265	1404	0.90	139	2086	2344	0.89	258
Other Race												
65–69	65	72	0.91	7	79	89	0.89	10	144	161	0.90	17
70–74	53	56	0.95	3	59	66	0.90	7	113	122	0.92	9
75–79	36	39	0.91	3	31	46	0.67	15	66	85	0.78	19
80–84	20	19	1.05	−1	18	26	0.70	8	38	45	0.84	7
85+	11	13	0.81	2	14	19	0.75	5	25	32	0.77	7
Total	185	199	0.93	14	201	246	0.82	45	386	445	0.87	59
Unknown/missing race												
65–69	118	0	—	−118	155	0	—	−155	273	0	—	−273
70–74	97	0	—	−97	130	0	—	−130	227	0	—	−227
75–79	62	0	—	−62	106	0	—	−106	168	0	—	−168
80–84	28	0	—	−28	55	0	—	−55	83	0	—	−83
85+	16	0	—	−16	57	0	—	−57	74	0	—	−74
Total	322	0	—	−322	503	0	—	−503	825	0	—	−825
All Races												
65–69	3895	4254	0.92	359	4803	5176	0.93	373	8698	9430	0.92	732
70–74	3086	3213	0.96	127	4194	4353	0.96	159	7281	7566	0.96	285
75–79	2047	2135	0.96	88	3268	3360	0.97	92	5315	5495	0.97	180
80–84	1156	1154	1.00	−2	2235	2176	1.03	−59	3391	3330	1.02	−61
85+	766	773	0.99	7	1978	1938	1.02	−40	2743	2711	1.01	−32
Total	10951	11529	0.95	578	16478	17003	0.97	485	27428	28532	0.96	1104

Notes: Estimates of Medicare enrolled population calculated by taking average of live counts for 5% national sample for 12/31/84 and 12/31/85 in each relevant cell and multiplying by 20. Persons recorded as on the live HISKEW file but who were not residents of the 50 states were excluded. Census estimates were for 7/1/85, and were obtained from the Current Population Survey.[12]

HSK = Estimate of population from Medicare's HISKEW file.
CNS = Estimate of population from Current Population Survey.
H/C = Ratio of HISKEW estimate to CPS estimate.
C-H = Census estimate minus HISKEW estimate

aged 65 to 70 is enrolled in Medicare. Among Blacks, the proportion enrolled increases with age and exceeds the Census estimates among those over 80 years old. No notable differences between the two genders in the age-specific proportions enrolled in Medicare are apparent for Whites or Blacks. However, among those whose race is designated as "other," different patterns are apparent for males and females. Finally, for 3.1 percent of the individuals in the HISKEW file, race is designated as unknown, while the Census assigns a race designation to all individuals.

Case Ascertainment from Hospital Claims

Table 2 presents data on the proportion of hip fracture cases identified from the Part B (physician and other provider) files by state for 1985. Overall, 9.1 percent of hip fracture cases were identified from the Part B claims alone, and the proportion of cases so identified ranged from none in several states to a high of 32.8 percent in Virginia. The proportion of cases identified only through the Part B claims decreased significantly with age from 10.6 percent for those ages 65 to 69 to 8.4 percent for those age 85 and over (Chi square, 4df, 12.9, p = .01). However, no significant differences were found in the proportion of cases identified through the Part B files by gender or race.

Discussion

It is often assumed that the Medicare-eligible population corresponds closely to the elderly population of the United States and that studies of Medicare's hospital discharges will provide accurate estimates of hospital utilization among the elderly. One recent study has estimated hip fracture incidence rates using data from the US Census to estimate age/race/gender-specific denominators, while using Medicare hospital discharge data to calculate age, race, and gender-specific numerators for these rates.[3] Our data suggest that this approach must be taken with considerable caution.

We found that the Medicare population over the age of 65 differs in several important ways from the US Census estimates of the elderly population as a whole. The fact that only 95 to 96 percent of US elderly residents are enrolled in Medicare has been previously reported,[14] but breakdowns by age, gender, or race were not provided. Our data demonstrate that Medicare coverage varies substantially across these groups. Although some of the differences between Census and Medicare estimates of the population are trivial, others

TABLE 2—Proportion of Hip Fracture Cases Identified through the Medicare Part B Claims only, by State, based on 5% National Sample, Calendar Year 1985

State/Region	N Cases	% B Only
NORTHEAST		
Connecticut	128	0.0
Maine	46	4.3
Massachusetts	249	13.3
New Hampshire	38	7.9
New Jersey	276	18.1
New York	685	11.1
Pennsylvania	479	7.9
Rhode Island	48	12.5
Vermont	19	0.0
Region Total	1968	10.6
MIDWEST		
Illinois	439	11.4
Indiana	262	7.6
Iowa	156	4.5
Kansas	132	3.0
Michigan	268	9.7
Minnesota	151	4.6
Missouri	254	4.7
Nebraska	63	4.8
North Dakota	33	9.1
Ohio	443	11.5
South Dakota	32	3.1
Wisconsin	208	2.4
Region Total	2441	7.7
SOUTH		
Alabama	141	10.6
Arkansas	109	5.5
Delaware	27	3.7
District of Columbia	9	11.1
Florida	522	11.7
Georgia	186	7.5
Kentucky	159	6.9
Louisiana	99	4.0
Maryland	111	9.9
Mississippi	78	7.7
North Carolina	179	27.4
Oklahoma	146	4.8
South Carolina	68	5.9
Tennessee	195	4.6
Texas	521	7.5
Virginia	131	32.8
West Virginia	78	6.4
Region Total	2759	10.4
WEST		
Alaska	3	0.0
Arizona	107	5.6
California	821	8.6
Colorado	107	9.3
Hawaii	10	0.0
Idaho	33	3.0
Montana	29	3.4
Nevada	24	0.0
New Mexico	43	2.3
Oregon	123	5.7
Utah	27	0.0
Washington	163	6.7
Wyoming	15	6.7
Region Total	1505	7.2

are not. Consideration of sampling variations would further complicate interpretation of these differences. However, our analyses underline the obvious but important point that Medicare data provide information about the Medicare population; findings of studies that rely on Medicare data should be generalized to the elderly population as a whole with caution.

A full exploration of the causes of these differences is beyond the scope of this paper but would include issues related to criteria for Social Security eligibility, delayed enrollment for those still employed after age 65, and decisions by eligible persons about whether to enroll in either or both of the Medicare Part A and Part B programs. That the Current Population Estimates data from the US Census bureau are estimates subject to both sampling and measurement error may also play a role.

Equally important, coding conventions for race used by the Census and by Medicare differ. While the Census assigns a racial designation to all residents, approximately 3 percent of Medicare enrollees remain of unknown race. Given the White predominance in the elderly population and the possibility that substantial numbers of Whites are included in the group designated unknown, it would be inappropriate to group those of unknown race with either of the other two minority groups.

Finally, we must note that we cannot explain the relative excess of Medicare eligibles compared to Census estimates for the older age groups in several racial categories. Although Census undercounts in the oldest population are conceivable, this could also be explained by the failure to record a small number of deaths in the HISKEW file, a deficiency that would be magnified in the oldest age categories. We have found some indication that this might be the case. The 1985 5 percent national HISKEW file included 368 individuals over the age of 110, resulting in an estimated 7,360 individuals over this age in the population as a whole—an implausible figure. Nevertheless, given the close similarity of our overall estimates of the Medicare population based on HISKEW to HCFA's estimate of the proportion of the US population enrolled in Medicare (95 percent[14]), few deaths overall are apparently missed by these files and analytic difficulties caused by this problem would be serious only among the oldest old.

While it is obvious that outpatient claims would be required to analyze incidence rates for fractures that can be treated in an outpatient setting, we found that Medicare's hospital discharge files do not capture all treatment for hip fractures, a condition virtually always treated on an inpatient basis. Overall, physician claims were required to identify 9.1 percent of hip fracture cases; almost all of these physician claims were coded with the place of service "inpatient." For the majority of these cases, we found associated hospital claims with evidence of treatment for orthopedic conditions related to the hip, but no diagnosis of hip fracture. For the remainder, we must conclude that the hospital claims were either lost in the data transmission process or severely miscoded. It is reassuring that only small and insubstantial differences in fracture detection rates were found by age, race or gender, but the variation by state of residence might pose problems for analyses of geographic differences in incidence rates across states. We should also note that gaps in the physician claims are possible—Connecticut Part B claims were missing from the 1985 Medicare Part B 5 percent national file. A second caveat is that some Medicare enrollees may receive care in Veterans' Administration hospitals. We found that approximately 4 percent of New England hip fracture cases among males (and less than 1 percent among females) were documented in VA files.[13]

Our data illustrate two specific limitations beyond the accuracy of diagnostic coding that must be considered when using or reporting findings of studies that rely on Medicare data. First, the Medicare population cannot be assumed to correspond exactly with the resident US elderly population. Second, gaps in the Medicare hospital data must be acknowledged. These potential limitations could also apply to any analysis where different data sources are used for the numerator and denominator, or where hospital discharge data sets are not complete prior to release as public use tapes.

Most important, however, our data underline the feasibility of overcoming these limitations through the careful linkage of data from the multiple files available through the Medicare program. One of the cardinal rules in calculating rates is to ensure that the criteria used to determine eligibility for inclusion in the numerator should be as similar as possible to those used for assignment to the denominator. The Medicare enrollment file (HISKEW) can be used not only to define precisely denominators by age, race, sex, and mailing address (zip code), but can also be used to define identical inclusion criteria for both the numerator and denominator of rates. Finally, physician claims obtained from the Part B files and hospital claims from the Veterans' Administration can be merged with the Medicare hospital claims files to reduce the likelihood of missing cases in the study population of interest. When the limitations of administrative health care data bases are recognized and appropriate care exercised in their use, they can provide a valuable tool for epidemiologic research.

REFERENCES

1. Silverman SL, Madison RE: Decreased incidence of hip fracture in Hispanics, Asians and Blacks: California hospital discharge data. Am J Public Health 1988; 78:1482–1483.
2. Farmer ME, White LR, Brody JA, Bailey KT: Race and sex differences in hip fracture incidence. Am J Public Health 1984; 74:1374–1380.
3. Kellie SE, Brody JA: Sex-specific and race-specific hip fracture rates. Am J Public Health 1990; 80:326–328.
4. McPherson K, Wennberg JE, Hovind OB, Clifford P: Small-area variations in the use of common surgical procedures: An international comparison of New England, England, and Norway. N Engl J Med 1982; 307:1310–1314.
5. Wennberg JE, Freeman JL, Shelton RM, Bubolz TA: Hospital use and mortality among Medicare beneficiaries in Boston and New Haven. N Engl J Med 1989; 321:1168–1173.
6. Health Care Financing Administration: Medicare Hospital Mortality Information 1987. Washington, DC: Govt Printing Office, 1988.
7. Roper WL, Winkenwerder W, Hackbarth GM, Krakauer H: Effectiveness in health care. An initiative to evaluate and improve medical practice. N Engl J Med 1988; 319:1197–1202.
8. Roos NP, Wennberg JE, Malenka DJ, *et al*: Mortality and reoperation after open and transurethral resection of the prostate for benign prostatic hyperplasia. N Engl J Med 1989; 320:1120–1124.
9. Jencks SF, Williams DK, Kay TL: Assessing hospital-associated deaths from discharge data. The role of length of stay and comorbidities. JAMA 1988; 260:2240–2246.
10. Greenfield S, Aronow HU, Elashoff RM, Watanabe D: Flaws in mortality data. The hazards of ignoring comorbid disease. JAMA 1988; 260:2253–2255.
11. Iezzoni LI, Burnside S, Sickles L, Moskowitz MA, Sawitz E, Levine PA: Coding of acute myocardial infarction: clinical and policy implications. Ann Intern Med 1988; 109:745–751.
12. US Bureau of the Census, Department of Commerce, Current Population Reports, Series P-25, No. 985, Table 3. Estimates of the population of the US by age, race and sex, 1980–1985. Washington, DC: Govt Printing Office, 1986.
13. Fisher ES, Baron JA, Malenka DJ, Barrett JA, Kniffin WD, Whaley FS, Bubolz TA: Hip fracture incidence and mortality in New England. Epidemiology (In press).
14. Hatten J: Medicare's common denominator: The covered population. Health Care Financ Rev 1980; 2:53–63.

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Prospective Payment and the Utilization of Physical Therapy Service in the Hospitalized Elderly

PETER HOLT, MD, AND CAROL HUTNER WINOGRAD, MD

Abstract: We studied the effect, in a university teaching hospital, of the prospective payment system (PPS) on utilization of physical therapy (PT), a non-reimbursable service; subjects were hospitalized patients aged 75 or older with non-PT-related diagnoses (myocardial infarction, pneumonia, congestive heart failure, and colectomy) and PT-related diagnoses (cerebrovascular accident and hip fracture). The proportion of patients referred for PT increased from 68 percent pre-PPS to 85 percent post-PPS for those with PT-related diagnoses and from 13 percent pre-PPS to 19 percent post-PPS for those with non-PT-related diagnoses. The mean number of sessions of PT decreased slightly for both groups: from 8.5 to 7.6 sessions for those with PT-related diagnoses and from 5.2 to 4.5 for those with non-PT-related diagnoses. In patients with PT-related diagnoses whose ambulatory status worsened during hospitalization, referrals for PT increased from 76 percent pre-PPS to 98 percent post-PPS. Referrals of comparable patients with non-PT-related diagnoses did not increase. Changes in provider education and efforts to reduce length of stay may account for these findings. (*Am J Public Health* 1990; 80:1491–1494.)

Introduction

In 1983, the Health Care Financing Administration (HCFA) introduced the prospective payment system (PPS). PPS created an incentive for hospitals to provide care at a cost at or below the established amount for a specific diagnosis-related group (DRG). Cost reduction could be accomplished by either reducing length of stay (LOS) or by decreasing utilization of ancillary services, such as physical therapy (PT). Nationally, LOS has decreased since the implementation of PPS.[1–5] In contrast, little attention has been directed toward the impact of PPS on utilization of PT.

This study focused on PT because it is a service used by hospitalized patients with a broad range of diagnoses. In 1986, approximately one in six hospitalized patients received some type of PT treatment.* Second, PT is a clinical service designed to improve mobility, ambulation, and independence. Because ambulatory status affects both quality of life and discharge status, the accessibility to and availability of PT is important, particularly to the frail elderly.[6–8]

Previous studies examining the effects of PPS on utilization of hospital PT services have shown mixed results. Several studies revealed a significant decrease in the mean number of PT sessions in hip fracture patients treated after the implementation of PPS.[2,5,9] These studies, however, did not evaluate the percentage of patients referred for PT. Another study found a significant increase in referrals for inpatient PT since 1983 in small acute care rural hospitals.[10] In contrast, a more recent study from 501 non-federal acute care hospitals reported a decrease in the number of patients referred for PT since 1983.[11] Neither of the latter two studies addressed which categories of patients (i.e. which DRGs) were more likely to have experienced changes in the pattern of utilization of PT.

The current study was designed to document the utilization patterns of PT services in the elderly prior to and after the implementation of PPS via two different indicators of PT utilization: percentage of referrals and mean number of sessions.

Methods

Medical records of patients 75 years of age or older discharged from Stanford University Medical Center (SUMC) were examined retrospectively for six consecutive years from September 1, 1980 through August 31, 1986. This represented four years prior to the implementation of PPS (9/1/80–8/31/84) and two years after the implementation of PPS (9/1/84–8/31/86).

Subjects

All patients discharged with one of the following six principal conditions were eligible: cerebrovascular accident (CVA); pneumonia; myocardial infarction (MI); congestive heart failure (CHF); hip fracture; and colectomy. These were further categorized into two groups: PT-related diagnoses which included CVA and hip fracture, and non-PT-related diagnoses which included pneumonia, MI, CHF, and colectomy. MI was not included in PT-related diagnoses because Medicare does not recognize it as a "rehabilitation diagnosis" in spite of the proliferation of cardiac rehabilitation programs.

After PPS was implemented, there was an incentive for chart abstractors in the Medical Records Department to be as specific as possible when coding. In the two years post-PPS, fewer patients than pre-PPS were discharged with CVA. Instead they were categorized with the specific etiology of CVA (e.g. embolic, thrombotic, or hemorrhagic) which resulted in a different ICD code. All other categories remained the same pre- and post-PPS.

The study sample (n = 917) was selected from a list of patients by use of tables of random permutations.[12] Because of the small numbers in the CVA category for the post-PPS years, all eligible patients were included.

Data Collection and Analysis

Data were abstracted from the medical records with the number of PT sessions counted only if the patient actually received therapy. The initial PT evaluation was counted as one session. The percentage of referrals and the mean number of sessions were calculated.

From St. Luke's Hospital, Kansas City Missouri (Holt); Department of Medicine, Division of Gerontology, Stanford University School of Medicine, California (Winograd); and the Veterans Affairs Medical Center, Palo Alto, California (Holt, Winograd). Address reprint requests to Peter Holt, MD, St. Luke's Hospital, 44th and Wornall Road, Kansas City, MO 64111. This paper, submitted to the Journal May 15, 1989, was revised and accepted for publication May 29, 1990.

*Terbush TW: Estimates of physical therapy costs and usage by Medicare patients in FY 1986. Prepared for the American Physical Therapy Association, January 1987.

Ambulatory status was categorized as follows: unable to walk, walks only with the physical assistance of another person, ambulates independently with or without ambulation aids, or not obtainable. It was determined just prior to the admission illness or injury (i.e. pre-morbid ambulatory status) and at discharge. For those patients in whom ambulatory status was obtainable at both times, the difference between pre-morbid ambulatory status and discharge ambulatory status was scored better, worse, or the same.

Residence at admission and discharge was categorized as follows: independent home setting, board and care home, acute care facility, intermediate care facility (ICF), skilled nursing facility (SNF), or other type of facility. Any change in type of residence was recorded.

The predictor variable was relationship to PPS, divided into pre-PPS and post-PPS. The outcome variables were utilization of PT (measured by mean number of PT sessions and percent of patients referred to PT), length of stay, change in ambulatory status, and change in type of residence.

Results were analyzed using t-tests for the continuous outcome variables and chi-square tests for dichotomous outcome variables. Ninety-five percent confidence intervals were calculated for all results.[13]

Results

We identified 917 patient charts for review after random selection. Of these, 907 (99%) were available for review, 468 (98%) of the 475 cases in the pre-PPS and 439 (99%) of the 442 cases in the post-PPS period. The two groups were similar in terms of age, sex, marital status, type of residence, and in-hospital mortality (Table 1). Length of stay decreased by 1.6 days.

The proportion of patients referred for PT increased from 68 percent pre-PPS to 85 percent post-PPS (diff = 17%, CI = 7.4, 26.6%) for the PT-related diagnoses group and from 13 percent pre-PPS to 19 percent post-PPS (diff = 6%, CI = 0.3, 11.7%) for the non-PT-related diagnoses group. However, the mean number of PT sessions decreased slightly from 8.5 sessions pre-PPS to 7.6 sessions post-PPS (diff = 0.9, CI = −0.8, 2.6%) for the PT-related diagnoses group and from 5.2 sessions pre-PPS to 4.5 sessions post-PPS (diff = 0.7, CI = −0.7, 1.4%) sessions post-PPS for the non-PT-related diagnoses group (Table 2).

Patients with PT-related diagnoses whose ambulatory status worsened were referred for PT more often post-PPS (76% pre-PPS to 98% post-PPS, diff = 22%, CI = 9.5, 34.5%), while PT referrals of comparable patients with non-PT-related diagnoses showed no change. Discharge to a more dependent level of care increased for patients with PT-related diagnoses (44% pre-PPS to 59% post-PPS, diff = 15%, CI = 2, 28%), while no difference existed in the group with non-PT-related diagnoses.

TABLE 1—Demographic Data of Subjects Pre- and Post-Prospective Payment System

Item	Pre-PPS (N = 468)	Post-PPS (N = 439)	Difference (95% CI)
Age (Mean)	82.5	83.0	0.5 (−0.2, +1.2)
Sex (% Male)	38.0	38.0	0
Marital Status (% Married)	38.0	38.0	0
Residence (% Home)	86.0	84.0	2 (−13, +17)
Mortality (%)	16.0	13.0	3 (−11, +17)
Length of Stay (Number of Days)	10.7	9.1	1.6 (+0.7, +2.5)

The proportion of patients ambulating independently prior to admission did not change for the group with PT-related diagnoses however, there was a small decrease at discharge (54% pre-PPS to 46% post-PPS, diff = −8%, CI = −5, 21%). The proportion of patients in the non-PT-related diagnoses group that ambulated independently decreased prior to admission (95% pre-PPS to 90% post-PPS, diff = −5%, CI = 0.2, 9.8%) and at discharge (87% pre-PPS to 80% post-PPS, diff = −7%, CI = 0.03, 13.7%) (Table 3).

Discussion

This study documents an increase in the percentage of patients referred for PT after the implementation of PPS in a university hospital. This is a surprising and encouraging finding, because prior to the study we expected to find a decrease in utilization of PT services. Our findings do not substantiate previous concerns that the elderly would inevitably suffer from poorer quality of care under PPS, as hospitals under pressure to maintain their "bottom line" would reduce the provision of potentially beneficial services.

A small decrease occurred in the mean number of PT sessions administered in the post-PPS time period. This decrease was less than one might have expected given the considerable decrease in the LOS; this contrasts with the results of other investigators who found decreases in the LOS and mean number of PT sessions to be more closely related.[2,5,9]

Physicians, who must initiate the referral for PT, may have become more aware of the benefits of early mobilization and ambulation during acute illness; referral for PT services may help maintain mobility and thereby enable patients to be discharged sooner.

The overall ambulatory status of patients appears to have declined during the period of implementation of PPS. After PPS, patients with non-PT-related diagnoses (e.g. CHF and colectomy) were more dependent in ambulation both prior to admission and at discharge. These patients were admitted with medical and surgical conditions that in themselves did not directly affect neurological or musculoskeletal function and should not have had an impact on ambulation. This dependency in ambulation may reflect the findings of other investigators that the case mix of patients after PPS changed.[14] Acute care hospitals now provide care to more severely ill patients in need of the greatest intensity of service. For example, after PPS, patients with mild forms of pneumonia and CHF may have been treated as outpatients, while only the most debilitated patients were admitted to the hospital.

Since one of the primary indications for and goals of PT is to maintain ambulation, we also examined the change in ambulatory status of patients admitted before and after PPS, and the referral patterns for PT in these patients. Although no difference occurred in the percentage of patients whose ambulatory status worsened during the hospitalization before or after PPS, subgroup analysis revealed striking differences between PT-related and non-PT-related groups in referral for PT. Patients with strokes and hip fracture whose ambulation worsened were more frequently referred for PT after PPS. In contrast, comparable patients with medical and surgical diagnoses experienced no increase in referrals for PT after PPS.

TABLE 2—Utilization of Physical Therapy Services Pre- and Post-Prospective Payment System by Diagnostic Group

	PT-Related Diagnoses			Non-PT-Related Diagnoses		
	Pre-PPS	Post-PPS	Difference (95% CI)	Pre-PPS	Post-PPS	Difference (95% CI)
Percent Referred	68	85	+17 (+7.4, +26.6)	13	19	+6 (+0.3, 11.7)
Mean Number of PT Sessions	8.5	7.6	−0.9 (−0.8, +2.6)	5.2	4.5	−0.7 (−0.7, +1.4)

TABLE 3—Percent of Patients Ambulating Independently Pre- and Post-Prospective Payment System by Diagnostic Group

	PT-Related Diagnoses			Non-PT-Related Diagnoses		
	Pre-PPS	Post-PPS	Difference (95% CI)	Pre-PPS	Post-PPS	Difference (95% CI)
Pre-Morbid (Prior to Illness Causing Hospitalization)	90	90	0	95	90	−5 (+0.2, +9.8)
At Discharge	54	46	−8 (−5, +21)	87	80	−7 (+.03, +13.7)
Pre-Post Difference	−36	−44	+8 (−5, +21)	−8	−10	+2 (+3, +7)

Impaired mobility is a common problem in the elderly[15] that is frequently exacerbated by acute care hospitalization.** Simple bedrest, a common order on admission for acutely ill patients, can be devastating in the frail elderly.[16–18]

We reviewed a random selection of 12 patient charts from the non-PT-related group with a decline in ambulatory status, but were not referred for PT services. No consistent factor (e.g. patient refusal, severity of illness, physician preference) could be identified to explain the absence of a PT request. Referrals may be based on different standards of practice among groups of physicians. Neurologists and orthopedists may be more familiar with rehabilitation than are internists. One would hope that patients with impaired mobility regardless of their diagnosis would receive the necessary PT services prior to discharge.

An encouraging finding in our study was that the percentage of patients with non-PT-related diagnoses requiring placement in a more dependent level of care did not increase after PPS. This is corroborated by other investigators.[19,20] However, more patients in the PT-related group (e.g. hip fractures) required a more dependent placement after PPS. Several studies have reported similar findings;[2,5,9] change in case mix, as well as a decreased LOS may explain them. After PPS, patients with hip fractures and strokes may not have had adequate recovery time in the hospital to ambulate well enough to go back to their original type of residence.

We do not think the decrease in the number of patients with CVA diagnostic code, after the implementation of PPS, affected the comparability of the two groups because the change in coding specified only the type of stroke and not the location or associated functional disability. Both groups most likely had an equal need for utilization of PT services.

In 1984, the Palo Alto Veterans Administration Medical Center, a major teaching hospital for Stanford University Medical Center housestaff, established a geriatrics program with a strong rehabilitation focus for acutely ill, hospitalized elderly veterans. Many members of the housestaff rotated on this service to learn the principles of geriatric medicine. These concepts may have been integrated into their treatment plans when the housestaff returned to Stanford Medical Center for subsequent rotations. This may have affected PT referrals.

A home health care program was introduced at Stanford during the study period. Greater availability of in-house support services, including health aides, nurses, and rehabilitation therapists, may have had an impact on discharge planning and discharge location.

In summary, this study describes a changing pattern of utilization of PT since the implementation of PPS in a university hospital. Specifically, the percent of patients referred for PT increased. In spite of this finding, this study also suggests that patients with medical and surgical diagnoses who have impaired ambulation may not be referred for potentially beneficial PT services.

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REFERENCES

1. Smith DB, Pickard R: Evaluation of the impact of Medicare and Medicaid prospective payment on utilization of Philadelphia area hospitals. Health Serv Res 1986; 21:529–546.
2. Fitzgerald JF, Fagan LF, Tierney WM, *et al*: Changing pattern of hip fracture care before and after implementation of the prospective payment system. JAMA 1987; 258:218–221.

**Hirsch C, Sommers L, Olsen A, *et al*: Discharged quick and functionally sick; functional morbidity among hospitalized elderly (Abstract). Anaheim, CA: American Geriatrics Society Annual Meeting, 1988.

3. Beebe K, Callahan W, Mariano A: Medicare short-stay hospital length of stay, fiscal years 1981–85. Health Care Financ Rev 1986; 7:119–125.
4. Guterman S, Eggers PW, Riley G, *et al*: The first three years of Medicare prospective payment: An overview. Health Care Financ Rev 1988; 967–976.
5. Gerety MB, Soderholm-Difatte V, Winograd CH: Impact of Prospective Payment and Discharge Location on the Outcome of Hip Fracture. J Gen Intern Med 1989; 4:388–391.
6. Tobis JS: Rehabilitation of the geriatric patient. *In*: Rossman I (ed): Clinical Geriatrics, 2d Ed. Philadelphia: J.B. Lippincott, 1979.
7. Applegate WB, Akins DE, Elam JT: A geriatric assessment and rehabilitation unit in a rehabilitation hospital. Clin Geriatr Med 1987; 3:145–154.
8. Liem PH, Chernoff R, Carter WJ: Geriatric rehabilitation unit: A three year outcome evaluation. J Gerontol 1986; 41:44–50.
9. Fitzgerald JF, Moore PS, Dittus RS: The care of elderly patients with hip fracture. Changes since implementation of the prospective payment system. N Engl J Med 1988; 1392–1397.
10. Dore D: Effects of Medicare prospective payment system on the utilization of physical therapy. Phys Ther 1987; 67:964–966.
11. Sloan FA, Morrisey MA, Valvona J: Medicare prospective payment and the use of medical technologies in hospitals. Med Care 1988; 26:837–853.
12. Moses L, Oakford RV: Tables of Random Permutations. Stanford: Stanford University Press, 1963.
13. Gardner MJ, Altman DG: Confidence intervals rather than P values: estimation rather than hypothesis testing. Br Med J 1986; 292:746–750.
14. Sloan FA, Morrisey MA, Valvona J: Case shifting and the Medicare prospective payment system.
15. Pinholt EM, Kroenke K, Hanley JF, *et al*: Functional assessment of the elderly. Arch Intern Med 1987; 147:484–488.
16. Policoff LD: The rehabilitation of the aged disabled. Soc NJ 1973; 70:586–588.
17. Strax TE, Ledebur JC: Rehabilitation in the elderly. *In*: Basmajian JV, Kirby RL (ed): Medical Rehabilitation. Baltimore: Williams and Wilkins, 1984.
18. Harper CM, Lyles YM: Physiology and complications of bedrest. J Am Geriatr Soc 1988; 36:1047–1054.
19. DesHarnais S, Kobrinski E, Chesney J, *et al*: The early effects of the prospective payment system on inpatient utilization and the quality of care. Inquiry 1987; 24:7–16.
20. Morrisey MA, Sloan FA, Valvona J: Medicare prospective payment and post-hospital transfers to subacute care. Med Care 1988; 26:685–698.

Conference Proceedings Available on Primary Care Research

As part of its emphasis on primary care for patients of all ages and conditions, the Agency for Health Care Policy and Research (AHCPR), in cooperation with several other agencies, convened a conference entitled "Primary Care Research: An Agenda for the 90s" in March 1990 at Colorado Springs, CO.

The conference explored many of the emerging issues expected to set the pace for primary care research during the 1990s, including: changing provider behavior, research with disadvantaged populations, medical treatment effectiveness in primary care mental health, community-oriented primary care, prevention research, outcome measurement issues, rural health, AIDS, substance abuse, practice-based research networks, and the federal perspective on primary care research.

These and other topics are presented in feature articles and commentaries in a new 147-page report resulting from the conference.

Single copies of the proceedings, *Primary Care Research: An Agenda for the 90s* (DHHS Pub. No. PHS 90-3460) are available from: AHCPR, Publications and Information Branch, 18–12 Parklawn Bldg., Rockville, MD 20857; (301) 443-4100.

Public
Health Briefs

Prevalence of Chronic Bronchitis among US Hispanics from the Hispanic Health and Nutrition Examination Survey, 1982–84

KI MOON BANG, PHD, MPH, PETER J. GERGEN, MD, MPH, AND MARGARET CARROLL, MSPH

Abstract: In the Hispanic Health and Nutrition Examination Survey (HHANES), Puerto Ricans had a higher age-adjusted prevalence of self-reported chronic bronchitis (2.9 percent, 95% CI = 2.2, 3.6) than Mexican Americans (1.7 percent, 95% CI = 1.3, 2.1) or Cubans (1.7 percent, 95% CI = 0.9, 2.5). The prevalence of chronic bronchitis was at least 2 times higher in smokers as compared to nonsmokers among Puerto Ricans and Cubans, but not for Mexican Americans. (*Am J Public Health* 1990; 80:1495–1497.)

Introduction

Chronic bronchitis is part of chronic obstructive pulmonary disease (COPD), the fifth leading cause of mortality in the US. Mortality from COPD increased 43 percent between 1979 to 1986 (22.2/100,000 to 31.8/100,000).[1] The epidemiology of chronic bronchitis has been described in numerous studies of US Whites.[2–4] Little is known about the prevalence of chronic bronchitis among the US Hispanic population.

We present estimates of the prevalence of self-reported chronic bronchitis among the three Hispanic subgroups and the association between chronic bronchitis and measured risk factors among Hispanics based on the 1982-84 Hispanic Health and Nutrition Examination Survey (HHANES).

Methods

The HHANES was conducted by the National Center for Health Statistics from 1982 to 1984. The target population consisted of individuals ages 6 months–74 years in the three major Hispanic subgroups in selected areas of the United States: Mexican Americans in five Southwestern states (Arizona, California, Colorado, New Mexico, and Texas); Cubans in Dade County, Florida; and Puerto Ricans in the New York City area. The sample design of HHANES was a multistage, stratified, clustered sample of each of the Hispanic subgroups. Details of the HHANES have been published.[5]

In the household interview, respondents were asked two questions about chronic bronchitis: 1) "Has a doctor ever told you that you had chronic bronchitis?", 2) "Do you still have chronic bronchitis?" Positive responses to both questions were used to define chronic bronchitis.

All statistical analyses were carried out using programs compatible with the Statistical Analysis System.[6] Since HHANES was based on a complex survey design, sampling weights were used to estimate the prevalence of chronic bronchitis and complex samples variances were estimated by multiplying the simple random sample variance estimates by an average design effect.[7,8] To compare the prevalence of chronic bronchitis among the three Hispanic subgroups, estimates of chronic bronchitis were age-standardized to the distribution of the 1980 population. The trend for the age-specific prevalence was analyzed using the Armitage method.[9] The logistic regression analyses were performed using RTILOGIT program.[10] Odds ratios (ORs) and 95% confidence intervals (CI) of ORs are presented.[11]

Results

The response rates were 79.2 percent for Mexican Americans, 73.9 percent for Cuban Americans, and 80.3 percent for Puerto Ricans (Table 1). The level of nonresponse did not vary across age, race, sex, or income groups. The age distributions of the analytical sample were similar in the Mexican American and Puerto Rican samples, but the Cuban American sample was considerably older. The proportion of heavy smokers (> 20 cigarettes/day) among Puerto Rican and Cuban smokers was much higher than among Mexican American smokers (Table 1).

The age-adjusted prevalence of self-reported chronic bronchitis was 1.7 percent (95% CI: 1.3, 2.1) for Mexican Americans; 1.7 percent (95% CI: 0.9, 2.5) for Cuban Americans; and 2.9 percent (95% CI: 2.2, 3.6) for Puerto Ricans (Table 2); prevalence increased slightly with age in both males and females among Mexican Americans ($p < 0.001$). Failure to find a clear increase in prevalence with age among Cuban Americans and Puerto Ricans may be due to the smaller sample size of these groups.

To assess the role of measured risk factors of self-reported chronic bronchitis, logistic models were fitted using age, sex, smoking, and poverty status (Table 3). Former smokers were excluded from the analysis because of small numbers. In the model for Puerto Ricans, age (OR = 1.03, 95% CI = 1.01, 1.05), sex (OR = 2.69, 95% CI = 1.38, 5.26), and cigarette smoking (OR = 2.72, 95% CI = 1.57, 4.71) were significant risk factors for chronic bronchitis. However, only smoking (OR = 2.75, 95% CI = 1.14, 6.62) for Cuban Americans and age (OR = 1.03, 95% CI = 1.01, 1.05) for Mexican Americans were significantly associated with chronic bronchitis.

From the National Center for Health Statistics, Hyattsville, MD. Address reprint requests to Ki Moon Bang, PhD, MPH, Division of Health Examination Statistics, Room 900, National Center for Health Statistics, 6525 Belcrest Road, Hyattsville, MD 20782. This paper, submitted to the Journal February 20, 1990, was revised and accepted for publication May 29, 1990.

TABLE 1—Description of the Samples

Survey period	Mexican Americans July 1982–November 1983	Cuban Americans January–April 1984	Puerto Ricans May–December 198
Number of sample persons selected	6,868	1,874	2,748
Response rate (%)	79.2	73.9	80.3
Age Groups (%)			
12–34 yrs	60.1	37.9	56.8
35–54 yrs	26.1	34.8	28.0
55–74 yrs	13.8	27.3	15.2
Sex			
(%) Male	46.7	46.2	41.5
(%) Female	53.3	53.8	58.5
Cigarette smoking (%)			
Current smokers	27.2	26.3	29.8
Former smokers	14.8	17.4	12.6
Never smokers	58.0	56.3	57.6
% of heavy smokers (>20 cig./day)	23.8	58.3	50.0
Poverty income ratio (%)			
Below poverty (<1)	31.3	20.7	48.0
At/Above poverty (≥1)	68.7	79.3	52.0

TABLE 2—Age-specific Prevalence (%) of Chronic Bronchitis among Hispanic subgroups

	Males/Age (years)			Females/Age (years)			
	12–34	35–54	55–74	12–34	35–54	55–74	Total **
Mexican Americans							
Number	1,578	624	336	1,688	798	416	5,440
Prevalence	0.9	1.5	3.6	0.9	2.5	3.1	1.7
95% CI	0.7, 1.1	0.4, 2.7	1.8, 5.4	0, 1.8	1.4, 3.7	0.8, 5.4	1.3, 2.1
Cuban Americans							
Number	256	221	162	268	260	217	1,384
Prevalence	*	3.5	2.3	1.5	2.7	1.8	1.7
95% CI	*	0.3, 6.7	0, 5.3	0, 3.3	0.2, 5.2	0, 4.8	0.9, 2.5
Puerto Ricans							
Number	552	229	135	701	389	201	2,207
Prevalence	1.1	2.0	2.5	2.7	5.0	4.9	2.9
95% CI	0.2, 2.0	0, 4.1	0, 7.3	1.3, 4.1	2.5, 7.5	1.9, 7.9	2.2, 3.6

CI: Confidence interval.
*No reported chronic bronchitis.
**Age-adjusted prevalence.

Discussion

The high age-adjusted prevalence of chronic bronchitis among Puerto Ricans may have been due to the higher proportion of heavy cigarette smokers among Puerto Ricans and/or the high exposure to air pollution in New York City. Females had consistently higher age-adjusted prevalences of chronic bronchitis than males: in Puerto Ricans, 3.8 percent for males, 1.6 percent for females; in Mexican Americans, 1.8 percent for females, 1.6 percent for males; in Cuban Americans, 1.9 percent for females, 1.4 percent for males. A recent study on 2,111 Hispanics in New Mexico reported that the prevalence of chronic bronchitis among females was 4.3 percent as compared to 2.9 percent for males.[12] In the 1978 National Health Interview Survey, the prevalence of chronic bronchitis was higher in females (3.7 percent) than in males (2.9 percent).[13] A possible explanation for the female predominance might be that females visit clinics more often and are therefore aware of this diagnosis.[14] In contrast, some studies reported that the risk of chronic bronchitis was more than three-fold greater for men than women.[15]

The failure to find an association between smoking and chronic bronchitis in Mexican Americans may be due to the high proportion of light smokers. Other studies have shown cigarette smoking to be one of the most important risk factors for chronic bronchitis.[16,17] A recent study[18] found that heavy smokers had a six-fold increase in chronic bronchitis as

TABLE 3—Estimates of Odds Ratios with 95% Confidence Intervals of Risk Factors for Chronic Bronchitis[a]

	Odds Ratio	95% Confidence Interval
Mexican Americans		
Sex	1.09	0.66, 1.82
Age	1.03	1.01, 1.05
Smoking	1.04	0.61, 1.77
Poverty	0.84	0.50, 1.39
Cuban Americans		
Sex	1.28	0.53, 3.10
Age	1.02	1.00, 1.04
Smoking	2.75	1.14, 6.62
Poverty	0.61	0.24, 1.57
Puerto Ricans		
Sex	2.69	1.38, 5.26
Age	1.03	1.01, 1.05
Smoking	2.72	1.57, 4.71
Poverty	0.57	0.33, 0.99

Sex (1 = female, 0 = male), Age (continuous)
Smoking (1 = smokers, 0 = nonsmokers), Poverty (1 = below, 0 = at/above)
a) Past smokers excluded.

compared to nonsmokers. Other important risk factors of chronic bronchitis, not measured in HHANES, include occupational exposures,[19–21] airborne irritant gases,[22,23] viral or bacterial infection,[4,24] and familial aggregation.[25]

The American Thoracic Society's definition of chronic bronchitis requires a history of three months of cough and phlegm or sputum for more than two consecutive years.[26] This information was not collected during HHANES. The use of diagnosed cases only will underestimate the prevalence of chronic bronchitis. Other potential biases which may occur cannot be estimated in the data set but must be kept in mind when interpreting the results of this study.

In conclusion, our results show that chronic bronchitis was more prevalent in Puerto Ricans than Cuban Americans or Mexican Americans. Cigarette smoking was an important risk factor for chronic bronchitis among Puerto Ricans and Cuban Americans. Thus, smoking prevention and smoking cessation programs in the Hispanic population would help reduce the risk of chronic bronchitis.

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REFERENCES

1. National Center for Health Statistics: Vital Statistics of the United States, 1986. Vol II-Mortality Part A. US Department of Health and Human Services, Public Health Service, 1988. DHHS Pub. No. PHS 88-1122. Washington, DC: Govt Printing Office, 1988.
2. US Department of Health and Human Services: The Health Consequences of Smoking: Chronic obstructive lung disease. A report of the Surgeon General. DHHS Pub. No. PHS 84-50205. Rockville, MD: US Department of Health and Human Services, 1984.
3. Parkes WR: Occupational Lung Disorders, 2nd Ed. Boston: Butterworths, 1985; 17–22.
4. Higgins ITT: Epidemiology of chronic respiratory disease: A literature review. Pub. No. EPA-650/1-74-007. Washington, DC: US Environmental Protection Agency, 1974.
5. National Center for Health Statistics: Plan and operation of the Hispanic Health and Nutrition Examination Survey, 1982-84. Vital and Health Statistics, Series 1, No. 19 DHHS Pub. No. PHS 85-1321. Washington, DC: Govt Printing Office, September 1985.
6. SAS Institute: SAS user's guide. Cary, NC: SAS Institute, 1982.
7. Kovar MG: Approaches for the analysis of data. *In:* National Center for Health Statistics, Plan and Operation of the Hispanic Health and Nutrition Examination Survey, 1982-84. Vital and Health Statistics, Series 1, No. 19, DHHS Pub. No. PHS 85-1321. Washington, DC: Govt Printing Office, September 1985.
8. Shah BV: SESUDAAN: Standard errors program for computing of standardized rates from sample survey data. Research Triangle Park, NC: Research Triangle Institute, 1981.
9. Armitage, P: Statistical Methods in Medical Research, 2nd Ed. London: Blackwell Scientific Publication, 1973.
10. Shah BV, Folsom RE, Harrell FE, Dillard CN: Survey data analysis software for logistic regression. Columbus: Battelle Laboratories, 1984.
11. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research. Belmont, CA, Lifetime Learning Publications, 1982.
12. Samet JM, Coultas DB, Howard CA, Skipper BJ: Respiratory diseases and cigarette smoking in a Hispanic population in New Mexico. Am Rev Respir Dis 1988; 137:815–819.
13. US Department of Health and Human Services, Public Health Service: Report of task force on epidemiology of respiratory disease. Bethesda, MD: National Heart, Lung, and Blood Institute, NIH, 1981. Pub. No. 82-2019.
14. US Department of Health, Education, and Welfare, Public Health Service: Physician visits, United States, July 1966–June 1967. Hyattsville, MD: National Center for Health Statistics, 1968. DHEW Pub. No. 76-1299.
15. Tockman MS, Khoury MJ, Cohen BH: The epidemiology of COPD. *In:* Petty TL (ed): Chronic Obstructive Pulmonary Disease, 2nd Ed. New York: Marcel Dekker, 1985; 51–58.
16. Rom WN, Kanner RE, Renzetti AD, Shigeoka JW: Respiratory disease in Utah coal miners. Am Rev Respir Dis 1981; 123:372–377.
17. Lebowitz MD, Burrows B: Quantitative relationships between cigarette smoking and chronic productive cough. Int J Epidemiol 1977; 6:107–113.
18. Thurlbeck WM: Chronic airflow obstruction in lung disease. In: Bennington JL (ed) Major Problems in Pathology, Vol 5. Philadelphia: WB Saunders, 1976.
19. Holman CDJ, Psaila-Savona P, Roberts M, McNulty JC: Determinants of chronic bronchitis and lung dysfunction in Western Australian gold miners. Br J Ind Med 1987; 44:810–818.
20. Husman K, Koskenvuo M, Kaprio J, Terho ED, Vohlonen I: Role of environment in the development of chronic bronchitis. Eur J Respir Dis (Suppl) 1987; 152:57–63.
21. Brinkman GL, Block DL, Cress C: Effects of bronchitis and occupation on pulmonary ventilation over eleven year period. JOM 1972; 14:615–620.
22. Raj Pandey M: Domestic smoke pollution and chronic bronchitis in a rural community of the Hill Region of Nepal. Thorax 1984; 39:337–339.
23. Bouhuys A, Beck GJ, Schoenberg JB: Epidemiology of environmental lung disease. Yale J Biol Med 1979; 52:191–210.
24. Calder MA: The infective aetiology of chronic bronchitis. *In:* Simmonds FAH, Hunt LB (eds): Some Aspects of Chronic Bronchitis. Proceedings of a symposium held at the Royal Society of Medicine, London, L & S Livingstone, Ltd, 1963; 25–29.
25. Tager IA, Tishler PV, Rosner B: Studies of the familial aggregation of chronic bronchitis and obstructive airway disease. Int J Epidemiol 1978; 7:55–62.
26. American Thoracic Society: Definition and classification of chronic bronchitis, asthma, and pulmonary emphysema. Am Rev Respir Dis 1962; 35:762–768.

Prevalence and Restrictiveness of Smoking Policies in King County, Washington, Manufacturing Worksites

SUSAN KINNE, PHD

Abstract: This study examined prevalence of smoking policies and of significant smoking restrictions in a 1988 random sample of 1,528 manufacturing companies in the Seattle, Washington area, and related these characteristics to worksite size and industry type. Sixty-nine percent of companies had formal smoking policies and 85 percent regulated smoking to some degree. Workforce size was directly related to prevalence of significant smoking restrictions, but when type of industry was controlled, the linear trend in the size/prevalence relationship was sustained in only three of the 18 industry groupings. (*Am J Public Health* 1990; 80:1498–1500.)

Introduction

Policies regulating workplace smoking have been imposed to reduce fire hazard, protect equipment[1] and, most recently, to protect nonsmokers from exposure to environmental tobacco smoke.[2] Efforts to assess the effects of these policies on smoking reduction and cessation have been reported.[3-8] Little is known about policy prevalence and content, because most policy surveys have had nonrandom sampling techniques, undefined populations, and low response rates that limit the quality of the data.[9-11] Few studies have clearly distinguished among types of policies or investigated relationships between organizational characteristics and policy content.[12] This research was designed to provide an accurate assessment of the prevalence of degrees of policy restriction on smoking in a geographically defined population of organizations employing many smokers, and to describe the relationship between policy, organizational size, and industry type.

Methods

These data are from a random sample of all King County (Washington) manufacturing worksites, surveyed by telephone between November 1988 and July 1989. King County includes the city of Seattle. The sampling frame, the set of all companies with manufacturing Standard Industrial Codes (SIC),[13] and King County zip code mailing addresses, was identified by a commercial broker (n = 2,883). Two-thirds of these companies (n = 1,922) were randomly selected for the study. Three hundred ninety-four companies were deleted as ineligible (disconnected numbers; non-County or non-manufacturing concerns; and one-person companies) on grounds that such companies would not use policy to control behavior. The response rate among eligible companies was 91.3 percent (1,395/1,528).

Companies in the sample received a letter describing the study, followed by a 10-minute telephone interview with the company head or a person identified by the head as knowledgeable about smoking policy. The informant was asked, "Does your organization presently have a policy on where employees can and cannot smoke in your buildings and facilities?" Policy was defined as a directive on smoking by the company head or governing body, communicated to employees in written or unwritten form.

Policies vary in the degree to which they limit smoking. Table 1 gives the policy response categories in order of increasing restrictiveness. Worksites also can be grouped by their degree of policy-mandated protection against environmental tobacco smoke (ETS) and the inferred company attitude toward smoking. Companies with no policy, with non-restrictive policies, or with smoking allowed except in no-smoking areas offer little ETS protection and accept smoking as the norm.[14] These were labeled "Smoking Allowed." Companies in which nonsmoking is the norm[14] were grouped as "Significant Restrictions." These include worksites that limit smoking to designated smoking areas and those that ban smoking completely, thereby limiting ETS exposure substantially. The prevalence of "Significant Restrictions" was examined for worksites of differing workforce size and industry type.

TABLE 1—Distribution of Smoking Policy Restrictions by Number of Employees in King County (WA) Manufacturing Worksites

	Number of employees				Total
Policy Restrictiveness	2–10	11–25	26–100	>100	%/n
(n of companies in category)	(700)	(326)	(269)	(100)	(1395)
Smoking allowed					
No policy on smoking	19.7%	15.6	4.5	1.0	14.5/202
Policy allows smoking everywhere	0.0	0.6	0.4	1.0	0.3/4
Non-policy smoking restrictions	19.6	14.7	11.9	10.0	16.3/227
Smoking except in no-smoking areas	4.9	9.2	14.1	10.0	8.0/112
(subtotal: smoking allowed)	(44.1)	(40.2)	(30.9)	(22.0)	(39.1/545)
Significant restrictions					
Smoking only in designated areas	12.9	26.7	40.1	57.0	24.5/342
No smoking allowed	27.9	27.0	23.4	16.0	25.9/362
No smoking, prefer to hire nonsmokers	10.9	4.9	4.5	4.0	7.7/108
No smoking, only hire nonsmokers	4.3	1.2	1.1	1.0	2.7/38
(subtotal: significant restrictions)	(55.9)	(59.8)	(69.1)	(78.0)	(60.9/850)

Address reprint requests to Susan Kinne, PhD, Fred Hutchinson Cancer Research Center, MP-702, 1124 Columbia Street, Seattle, WA 98104. This paper, submitted to the Journal January 29, 1990, was revised and accepted for publication May 29, 1990.

TABLE 2—Relationship between Prevalence of Significant Smoking Restrictions and Workforce Size, Controlling for SIC Group, King County (WA) Manufacturing Worksites

Industry by SIC Group Number (ranked by mean N of employees per company)	N of companies in group	Prevalence of restrictions (%) by N of employees				Prevalence for Group (%)	χ^2 Test for Trend	(p)
		2–10	11–25	26–100	> 100			
37: Transportation equipment	85	41.7	46.2	57.9	76.5	52.9	5.396	.020
36: Electrical, electronic equiment	84	63.6	53.8	77.8	90.9	70.2	3.425	.064
20: Food and kindred products	75	58.8	87.5	76.2	76.9	76.0	.711	.399
22: Textile mill products	9	60.0	—	66.7	100.0	66.7	.333	.564
29: Petroleum refining	9	33.3	100.0	100.0	100.0	77.8	3.493	.062
26: Paper and allied products	32	60.0	77.8	85.7	83.3	75.0	1.538	.215
38: Measuring, photographic, clocks	46	65.4	77.8	88.9	100.0	73.9	2.661	.103
33: Primary metal industries	28	27.3	75.0	28.6	0.0	39.3	.015	.901
30: Rubber and plastic products	50	50.0	62.5	70.0	100.0	64.0	4.132	.042
23: Apparel and fabric products	50	77.3	53.8	46.2	100.0	64.0	2.053	.152
34: Fabricated metal products	129	34.5	27.3	64.5	28.6	39.5	3.090	.079
35: Machinery, except electrical	138	44.2	57.1	47.8	90.0	50.7	3.815	.051
39: Miscellaneous manufacturing	101	55.9	54.2	100.0	85.7	62.4	4.679	.031
24: Lumber and wood products	67	62.9	70.6	50.0	100.0	64.2	0.025	.875
25: Furniture and fixtures	44	57.1	53.8	75.0	100.0	61.4	1.002	.317
32: Stone, clay, glass, concrete	48	36.4	47.1	71.4	0.0	43.8	.827	.363
28: Chemicals and allied products	41	59.1	55.6	90.0	—	65.8	1.948	.163
27: Printing, publishing	355	65.2	65.8	76.0	75.0	67.0	1.492	.222
31: Leather goods	4		—	—	—	75.0	—	—
Total companies	1,395					60.9%		

Results

We found that 69.2 percent (966/1,395) of companies reported having a policy on where employees could smoke in buildings and facilities (Table 1). Four of these companies (0.3 percent) stated that they had adopted a smoking policy that explicitly permitted smoking in all parts of the worksite, leaving 68.9 percent (962/1,395) of companies with policies that restricted smoking. An additional 227 companies without smoking policies reported that smoking was not allowed in parts of their worksite. When the latter two categories were combined, the data show that smoking was limited to some degree in 85.2 percent (1,189/1,395) of responding worksites. Companies that banned smoking completely, with or without hiring policies on smokers, comprised 36.3 percent (508/1,395) of worksites surveyed. The prevalence of significant restrictions on smoking was 60.9 percent (850/1,395).

The number of full-time employees, a measure of company size, was related to smoking policy (Table 1). Having a formal smoking policy increased with workforce size, as did prevalence of significant smoking restrictions, although the likelihood of a total smoking ban was greatest in the smallest companies. Companies without smoking restrictions were in the minority in every size category.

This relationship between workforce size and prevalence of significant restrictions varied by type of manufacturing activity. When SIC grouping was controlled, the chi-squared test for trend for the relationship between size and prevalence was significant for only three of the 18 SIC groups (Table 2). Five groups showed weak size/prevalence relationships ($.05 < p < .10$), not all of which can be attributed to small numbers of cases. In the remaining 10 groups, the size/prevalence relationships were erratic, suggesting that factors peculiar to those industries may account in part for the distribution of significant smoking restrictions.

Discussion

The results of this survey demonstrate general acceptance of the concept of worksite smoking control among King County manufacturers of all sizes. The majority of employees in these industries now enjoy some protection from exposure to environmental tobacco smoke.

These data share some of the limitations of previous work on smoking policy. We have no measures of reliability or validity of the questions used to inquire about policy, and respondents' meaning of "a policy on smoking" may vary.[12] Although the survey data estimate the extent of policies, they provide no information on policy enforcement, making it impossible to estimate actual impact on air quality. Lastly, these data reflect practices of manufacturers in one geographic area, and may not be representative of this sector's smoking policies in other parts of the country. A number of prominent employers and local governmental units have "gone smoke-free" in King County in the past few years. Washington State, King County, and the City of Seattle impose relatively stringent restrictions on public smoking. None of these jurisdictions requires that private employers or worksite environments have policies (although such a law, exempting small worksites, has been proposed by the State Legislature), but public acceptance of smoking restriction is widespread. This attitude may explain the survey's finding that extensive control of smoking in the worksite may be accomplished without legislative mandate.

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REFERENCES

1. Walsh DC: Corporate smoking policies: a review and an analysis. JOM 1984; 26:17–22.
2. US Department of Health and Human Services: Reducing the health consequences of smoking: 25 years of progress. A Report of the Surgeon General. DHHS Pub. No. CDC 89-8411. Washington, DC: Govt Printing Office, January 1989.
3. Biener L, Abrams DB, Follick MJ, Dean L: A comparative evaluation of a restrictive smoking policy in a general hospital. Am J Public Health 1989; 79:192–195.
4. Hagey A: Implementation of a smoking policy in the US Army. NY State J Med 1989; 89:42–43.

5. Petersen LR, Helgerson SD, Gibbons CM, Calhoun CR, Ciacco KH, Pitchford KC: Employee smoking behavior changes and attitudes following a restrictive policy on worksite smoking in a large company. Public Health Rep 1988; 103:115–120.
6. Rosenstock IM, Stergachis A, Heaney C: Evaluation of smoking prohibition policy in a health maintenance organization. Am J Public Health 1986; 76:1014–1015.
7. Andrews JL: Reducing smoking in the hospital. Chest 1983; 84:206–209.
8. Rigotti NA, Hill Pikl B, Cleary P, Singer DE, Mulley AG: The impact of banning smoking on a hospital ward. Clin Res 1986; 34:833A.
9. Gottleib NH, Hedl JJ, Eriksen MP, Chan F: Smoking policies among private employers and public agencies in Texas: A statewide analysis. JNCI 1989; 81:200–204.
10. Walsh DC, McDougall V: Current policies regarding smoking in the workplace. Am J Ind Med 1988; 13:181–190.
11. US Department of Health and Human Services: The Health Consequences of Smoking: Cancer and chronic lung disease in the workplace. A Report of the Surgeon General. DHHS Pub. No. PHS 85-50207. Washington, DC: Govt Printing Office, 1985.
12. Rigotti NA: Trends in the adoption of smoking restrictions in public places and worksites. NY State J Med 1989; 89:19–26.
13. Scott WR: Organizations: Rational, Natural and Open Systems. Englewood Cliffs, NJ: Prentice-Hall, 1981; 30.
14. US Dept of Health and Human Services: The Health Consequences of Involuntary Smoking. A Report of the Surgeon General. DHHS Pub. No. PHS CDC 87-8398. Washington, DC: Govt Printing Office, 1985; 298.

Depressive Symptoms and Cigarette Smoking among Latinos in San Francisco

ELISEO J. PÉREZ-STABLE, MD, GERARDO MARÍN, PHD, BARBARA V. MARÍN, PHD, AND MITCHELL H. KATZ, MD

Abstract: We administered the Center for Epidemiological Studies Depression (CES-D) Scale to 547 San Francisco Latinos as part of a random digit dialing telephone survey to evaluate smoking behavior. Both men and women current smokers had the highest mean CES-D levels (9.7 and 14.3, respectively). Logistic regression analysis adjusting for gender, acculturation, education, age, and employment showed that current smokers had an odds ratio of 1.7 (95% CI = 1.3, 2.2) for significant depressive symptoms compared to former smokers (OR = 1.1; 95% CI = .8, 1.6) and never smokers (OR = 1). (*Am J Public Health* 1990; 80:1500–1502.)

Introduction

Cigarette smoking continues to be the most common cause of preventable morbidity and mortality in the US.[1] Three community studies[2–4] and a study of adolescents[5] have reported on possible relations between smoking status and depressive symptoms with conflicting results.

Five population-based surveys of Latino communities in California have reported a high rate of depressive symptoms compared to non-Latino White samples.[6–11] Female gender,[6,8,9] younger age,[6,9] formerly married status,[9–11] lower income,[6,10,11] less education,[9,10] unemployment,[11] and inability to speak English[9] have been associated with a higher level of depressive symptoms. Factors associated with increased depressive symptoms in the Hispanic Health and Nutrition Examination Survey were female gender, low educational achievement, low income, and US birth combined with Anglo-oriented acculturation.[12] To date, no study has examined smoking status as it relates to depressive symptoms among Latinos.

Although the overall prevalence of cigarette smoking among US Latinos is somewhat lower than that for Whites or Blacks, this rate reflects the substantially lower smoking proportions of Latino women compared to men.[13–15] Increasing acculturation to the mainstream US culture results in more smoking by Latino women, but less among Latino men, and thus may eventually narrow this smoking gender gap.[15]

We examined the possible relation of depressive symptoms, cigarette smoking status, and demographic variables, within a community survey of Latinos in San Francisco, part of an ongoing evaluation of a community intervention to promote smoking cessation.

Methods

Subjects were sampled from the 27 census tracts in San Francisco with at least 10 percent Latinos and representing two-thirds (N = 55,541) of all Latinos (N = 83,373) living in San Francisco according to the 1980 US Census.[16] Telephone prefixes corresponding to the census tracts were identified using a reverse telephone directory and the Mitofsky-Waksberg[17] method for random digit dialing was applied.[18] This method identifies nonworking and other ineligible prefixes in a first stage.

A household was eligible if, on the initial question, the person answering the telephone self-identified as Latino or if the majority of residents were Latino. Within a given household, the Latino adult between 18 and 65 years of age who had most recently celebrated a birthday was invited to respond. A structured questionnaire was administered anonymously by trained bilingual interviewers after subjects gave verbal consent. Participants responded in Spanish (69.7 percent) or in English (30.3 percent); 5.5 percent of eligible participants refused to be interviewed.

Depressive symptoms were measured using the Center for Epidemiological Studies Depression scale (CES-D)[19] which has been applied in Spanish in other studies with Latinos.[6–8,11] A CES-D score of 16 or more is considered to represent significant depressive symptoms, and validation studies with psychiatric and community samples showed that approximately one-third of persons scoring 16 or more on the CES-D are diagnosed with clinical depression.[20,21]

Never smokers were defined as persons who had smoked less than 100 cigarettes in their entire life. Gender, age,

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Address reprint requests to Eliseo J. Pérez-Stable, MD, Division of General Internal Medicine, Department of Medicine, University of California, 400 Parnassus Avenue, Room A-405, San Francisco, CA 94143-0320. Dr. B. Marín and Dr. Katz are with that same department; Dr. G. Marín is with the Department of Psychology, University of San Francisco. This paper, submitted to the Journal August 18, 1989, was revised and accepted for publication April 9, 1990.

education, employment in last two weeks, birthplace, and national background were asked, but marital status was not determined. A five-item acculturation scale with excellent reliability and validity was administered and scores were dichotomized into low (1 to <3) and high (≥ 3 to 5) acculturation groups.[22]

Data were analyzed using a standard statistical software package for a microcomputer (BMDP).[23] Variables that have been shown in previous research to be associated with depression were entered into a logistic regression model along with smoking status: acculturation (low/high), age (<30 / ≥30 years), employment status (yes/no), and education (<12 years / ≥12 years). The categorical variable smoking status was coded into two variables: current smoking (yes/no) and former smoking (yes/no).

Results

A total of 604 Latinos agreed to be interviewed by telephone between June and August 1988; 57 (9.4 percent) were excluded from this analysis because of incomplete information; their demographic characteristics and smoking status were similar to those completing the CES-D and acculturation scales for gender, age, acculturation, birthplace, education, and smoking status, but they were more likely to be unemployed. The 547 subjects completing the CES-D had a mean age of 35 years (SD = 12), 11 years (SD = 4.1) of education, and 59 percent were women. The mean duration of time living in the US for the foreign-born was 12.4 years (SD = 9.5) and the mean acculturation score for the entire sample was 2.4 (SD = 1.2). Overall smoking prevalence age-adjusted by the direct method[24] to the 1980 US Census[16] was 19 percent, with more men (29 percent) smoking than women (13 percent).

The overall mean level of depressive symptoms was 9.4 (95% CI = 8.6, 10.2), and 104 (19 percent) subjects scored 16 or greater on the CES-D. Table 1 shows the mean CES-D scores separately for men and women by demographic characteristics. Women reported a higher mean level of depressive symptoms than men for each category analyzed. Unemployment and less than high school education were also associated with a greater risk of depressive symptoms. Never smokers reported a lower mean level of depressive symptoms than current smokers or former smokers. The proportion of current smokers with a mean CES-D score ≥16 was 21.9 percent for men and 39.5 percent for women, compared to 11.8 percent and 18.5 percent, respectively, for never smokers.

The odds ratio (OR) of significant depressive symptoms (CES-D ≥16) by smoking status was evaluated using logistic regression analysis controlling for gender, acculturation, education, age, and employment status. Results, shown in Table 2, indicate that overall current smokers have a 70 percent greater risk of significant depressive symptoms than never smokers. The increased risk was present for both men and women. Former smokers do not have an increased overall risk of significant depressive symptoms although for women the risk is greater than for men.

Discussion

Among Latinos in San Francisco, current smokers have a modest increased risk of reporting significant depressive symptoms on the CES-D when compared to never smokers. Although unemployment and less years of education were associated with more depressive symptoms in this study, their effects were marginal compared to that of gender and smoking status. Marital status, social support and self-reported health status, which have been found to correlate with depressive symptoms in some studies,[9–11] were not determined as part of this survey and it is possible that these variables may partly account for our results.

Studies of personality profiles of smokers have generally found that smokers are more extroverted and that a modest

TABLE 1—Mean Depressive Symptoms by Demographic Variables among San Francisco Latinos

	Men			Women		
	N*	CES-D	% ≥ 16	N	CES-D	% ≥ 16
Total	225	8.0	14.2	322	10.4	22.4
Age (years)						
18–29	96	8.1	13.5	117	10.2	22.2
30–65	129	7.9	14.7	204	10.4	22.5
Acculturation score						
low	146	8.4	15.1	226	10.5	22.6
high	79	7.2	12.7	96	10.1	21.9
Birthplace						
Latin America	172	8.0	14.5	234	10.3	21.4
United States	52	8.2	13.5	87	10.5	25.3
National background						
Mexican American	86	8.2	16.3	92	11.1	25.0
Central American	109	8.0	12.8	183	9.6	20.0
Other Latinos	30	7.3	13.3	41	11.9	26.8
Employed last 2 weeks?						
yes	165	7.4	12.1	183	10.0	20.2
no	57	9.8	21.1	138	10.9	25.4
Education (years)						
0–11	73	9.3	19.2	136	11.9	26.5
≥12	150	7.3	12.0	183	9.3	19.7
Smoking status						
never	110	6.9	11.8	242	9.3	18.5
former	51	8.2	9.8	37	12.9	27.0
current	64	9.7	21.9	43	14.3	39.5

*Ns may not add up to total because of missing data.

TABLE 2—Adjusted[*] Odds Ratio of Significant Depressive Symptoms by Cigarette Smoking Status among San Francisco Latinos[†]

	Sample OR (95% CI%)	Men OR (95% CI)	Women OR (95% CI)
Never smokers[#]	1.0	1.0	1.0
Former smokers	1.1 (.8,1.6)	.8 (.5,1.5)	1.3 (.9,2.0)
Current smokers	1.7 (1.3,2.2)	1.4 (.9,2.2)	1.8 (1.2,2.5)

[*] Estimates were adjusted for acculturation level, education, age, and employment status. Estimates for the entire sample were also adjusted for gender.

[†] Significant depressive symptoms are defined as a CES-D score ≥16 (see text).

[#] Never smokers were the reference group.

relationship exists between smoking and neuroticism.[25] The few specific studies on smoking and depression have found contradictory results. In a survey of 1,003 adults living in Los Angeles in 1979, Frerichs and colleagues[2] also found a significantly higher level of depressive symptoms as measured by the CES-D among smokers compared to nonsmokers (10.02 vs 8.76). In a study of 2,018 British men evaluated with a scale for psychoneurotic profile, Haines, *et al*,[3] also found that scores on the depression subscale (and the other four) were higher for smokers than nonsmokers. However, in both the above mentioned studies, when confounding factors were controlled the association of smoking and depression was no longer significant. A community survey of 1,209 men and women in New Zealand, using a scale similar to the British study, did find that depression was significantly greater among heavy smokers when compared to never smokers even when controlling for confounding factors.[4] Finally, a sample of 50 patients undergoing elective cardiac catheterization in St. Louis were evaluated for clinical depression and cigarette smoking was the only predictor variable associated with a diagnosis of depression.[26]

Although the causality between feeling depressed and smoking cannot be assumed from these cross-sectional data, recent prospective studies support such a link. Depressed adolescents 15 to 16 years of age in New York public schools were found to be more likely to be heavy cigarette smokers than non-depressed adolescents at ages 24 to 25 years.[5] Other suggestive evidence is from a clinical trial of clonidine as a pharmacologic adjunct to smoking cessation in 71 heavy smokers, which showed that 61 percent had a history of major depression and that this history had a significant negative effect on cessation regardless of treatment.[27]

It is possible that smokers report more depressive symptoms than nonsmokers because they are more willing to admit negative characteristics about themselves. As smoking becomes a less socially acceptable behavior smokers may become more depressed as a consequence, thus confounding the association observed in this study. However, if these findings are confirmed in other studies, smoking cessation interventions may need to integrate depression prevention and treatment components to maximize effectiveness.

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REFERENCES

1. US Department of Health and Human Services: The Health Consequences of Smoking: Nicotine Addiction. Washington, DC: US Department of Health and Human Services, Public Health Service, Office on Smoking and Health 1988.
2. Frerichs RR, Aneshensel CS, Clark VA, Yokopenic P: Smoking and depression: A community study. Am J Public Health 1981; 71:637–640.
3. Haines AP, Imeson JD, Meade TW: Psychoneurotic profiles of smokers and non-smokers. Br Med J 1980; 280:1422.
4. Waal-Manning HJ, de Hamel FA: Smoking habit and psychometric scores: A community study. NZ Med J 1978; 88:188–191.
5. Kandel DB, Davies M: Adult sequelae of adolescent depressive symptoms. Arch Gen Psychiatry 1986; 43:255–262.
6. Frerichs RR, Aneshensel CS, Clark VA: Prevalence of depression in Los Angeles County. Am J Epidemiol 1981; 113:691–699.
7. Roberts RE: Prevalence of depressive symptoms among Mexican Americans. J Nerv Ment Dis 1981; 169:213–219.
8. Lang JG, Muñoz RF, Bernal G, Sorensen JL: Quality of life and psychological well-being in a bicultural Latino community. Hispanic J Behav Sci 1982; 4:433–450.
9. Vega WA, Warheit G, Buhl-Auth J, Meinhardt K: The prevalence of depressive symptoms among Mexican Americans and Anglos. Am J Epidemiol 1984; 120:592–607.
10. Vega WA, Kolody B, Valle R, Hough R: Depression symptoms and their correlates among immigrant Mexican women in the United States. Soc Sci Med 1986; 22:645–652.
11. Golding JM, Karno M: Gender differences in depressive symptoms among Mexican Americans and non-Hispanic Whites. Hispanic J Behav Sci 1988; 10:1–19.
12. Moscicki EK, Locke BZ, Rae DS, Boyd JH: Depressive symptoms among Mexican Americans: The Hispanic Health and Nutrition Examination Survey. Am J Epidemiol 1989; 130:348–360.
13. Marcus AC, Crane LA: Smoking behavior among US Latinos: An emerging challenge for public health. Am J Public Health 1985; 75:169–172.
14. Escobedo LG, Remington PL: Birth cohort analysis of prevalence of cigarette smoking among Hispanics in the United States. 1989; 261(1):66–69.
15. Marín G, Pérez-Stable EJ, Marín BV: Cigarette smoking among San Francisco Hispanics: The role of acculturation and gender. Am J Public Health 1989; 79:196–199.
16. Bureau of the Census, Department of Commerce: 1980 Census of Population. Age, Sex, Race, and Spanish Origin of the Population by Regions, Divisions and States, Washington, DC: Govt Printing Office, 1983.
17. Waksberg J: Sampling methods for random digit dialing. J Am Statist Assoc 1978; 73:40–46.
18. Marín G, Marín BV, Pérez-Stable EJ: Feasibility of a telephone survey to study a minority community: Hispanics in San Francisco. Am J Public Health 1990; 80:323–326.
19. Radloff LS: The CES-D scale: A self-report depression scale for research in the general population. Appl Psychol Measur 1977; 1:385–401.
20. Weissman MM, Sholomskas D, Pottenger M, *et al:* Assessing depressive symptoms in five psychiatric populations: A validation study. Am J Epidemiol 1977; 106:203–214.
21. Myers JK, Weissman MM: Use of a self-report symptom scale to detect depression in a community sample. Am J Psychol 1980; 137:1081–1084.
22. Marín G, Sabogal F, Marín BV, Otero-Sabogal R, Pérez-Stable EJ: Development of a short acculturation scale for Hispanics. Hispanic J Behav Sci 1987; 9:183–205.
23. Dixon W (ed): BMDP Statistical Software. Berkeley, CA: University of California Press, 1985.
24. Fleiss JL: Statistical Methods for Rates and Proportions. New York: John Wiley & Sons, 1981.
25. US Department of Health, Education, and Welfare, USPHS: Smoking and Health: A Report of the Surgeon General. Washington, DC: Govt Printing Office, 1979.
26. Carney RM, Rich MW, Tevelde A, Saini J, Clark K, Jaffe A: Major depressive disorder in coronary artery disease. Am J Cardiol 1987; 60:1273–1275.
27. Glassman AH, Stetner F, Walsh BT, Raizman PS, Fleiss JL, Cooper TB, Covey LS: Heavy smokers, smoking cessation and clonidine: Results of a double-blind, randomized trial. JAMA 1988; 259:2863–2866.

Dental Office Practices for Tobacco Users: Independent Practice and HMO Clinics

HERBERT H. SEVERSON, PHD, ELIZABETH G. EAKIN, BS, VICTOR J. STEVENS, PHD, AND EDWARD LICHTENSTEIN, PHD

Abstract: Surveys of independent dental practitioners and health maintenance organization (HMO) dentists and their adult male patients in Oregon found that cessation information and advice was being given to tobacco-using patients, especially smokeless tobacco users, and that the tobacco users surveyed expressed interest in obtaining help to quit. Oral health providers expressed an interest in further training in how to help their tobacco-using patients to quit. (*Am J Public Health* 1990; 80:1503–1505)

Introduction

In 1985 it was estimated that over 12 million Americans ages 12 and older had used chewing tobacco or snuff (smokeless tobacco) in the past year[1]; among males who use smokeless, 43 percent used it almost daily.[2] Studies have shown correlations between smokeless tobacco use and gingivitis, gingival recession, leukoplakia, and oral cancers.[3]

In 1978, the House of Delegates of the American Dental Association (ADA) resolved that ADA members be encouraged "to undertake an educational effort to inform their patients of the systemic and oral health hazards of chewing tobacco and snuff." A 1964 ADA resolution had encouraged its members to advise their patients about the hazards of cigarette smoking.

Recent articles have reviewed dentists' involvement in smoking cessation counseling.[4,5] However, we are not aware of similar information about dentists' involvement with smokeless tobacco prevention. In this paper we report the results of two surveys of dentists, dental hygienists, and their adult male patients.

Methods

Independent Practice (IP) Survey

An explanatory letter was mailed to all practicing members of the Lane County Oregon Dental Society. Of the 114 dentists, 24 (21 percent) refused to participate and two were used as pilot offices. The study was conducted over a two-week period in 1988 during which time questionnaires were to be given to all male patients over 18 years of age visiting the dental office. Questionnaires were also completed by dentists and hygienists. The final questionnaire return rate for dentists was 83 percent (73/88). Data were also received from 65 hygienists and 806 adult male patients. Neither the base population of hygienists nor the population of eligible male patients was known.

HMO Survey

This study was conducted in the Kaiser Permanente Dental Care Program based in Portland, Oregon, which provided comprehensive dental care to 85,000 enrolled members. Seventy full-time dentists and 65 full-time dental hygienists were employed in seven fully staffed dental offices. The questionnaire return rate was 61 percent for dentists and 68 percent for hygienists.

Overall, 1,506 of age-eligible male patients (56 percent of the male patient population) completed the questionnaire. Observers making spot checks in the clinics noted that receptionists often failed to give the questionnaire to patients, but of those who did receive questionnaires more than 90 percent returned them to the reception desk as requested.

Results

Table 1 presents tobacco use and attitudes toward provider advice from each sample.

Table 2 summarizes the reported interactions with smokeless tobacco users and smokers for dentists in both settings. Most offer some kind of information and cessation advice. All types of information and advice were given more often with smokeless tobacco-using patients than with smoking patients. However, only a minority of providers have cessation materials available in their offices. Dentists tend to be more optimistic and comfortable when advising about smokeless use than about cigarette smoking. The majority of dental professionals in both surveys were interested in learning about a smokeless tobacco cessation program. The data from hygienists generally paralleled those of dentists.

The majority of dentists report being pessimistic about the effectiveness of their advice or the likelihood of a patient attempting a cessation clinic program (Table 3). The majority of hygienists reported the need for further training and patient resistance to referral as the most important obstacles.

Discussion

The results show that dentists and hygienists in both settings are concerned about patient tobacco use and inter-

TABLE 1—Male Patient Tobacco Use and Attitude toward Provider Advice

	Independent Practice (IP) N = 80%	HMO Clinics N = 1506
% Using smokeless	6	3.9
% Smoking	18	26.9
% Smoking and smokeless	1.4	0.7
Smokeless users interested in recciving cessation assistance	41	39
Smokeless users who would consider advice	27	33
Smokeless users reporting cessation advice	44	not asked

Address reprint requests to Herbert H. Severson, PhD, Research Scientist, Oregon Research Institute, University of Oregon, 1899 Willamette Street, Suite 2, Eugene, OR 97401. Ms. Eakin is Research Assistant at the Institute; Dr. Stevens is Investigator at Kaiser Permanente Center for Health Research; Dr. Lichtenstein is Research Scientist and Professor at the Institute and the University of Oregon. This paper, submitted to the Journal March 30, 1990, was revised and accepted for publication May 15, 1990.

TABLE 2—Independent Practice and HMO Dentists Reported Interactions with Smokeless Tobacco Users and Smokers

	IP Dentists (N = 73)		HMO Dentists (N = 43)	
	ST Users	Smokers	ST Users	Smokers
Advice and information usually given				
Discuss health hazards (%)	88	55	77	47
Discuss benefits of quitting (%)	62	51	42	35
Advised cut down use (%)	26	18	23	21
Advised quit cold turkey (%)	30	11	30	7
Prescribe nicotine gum (%)	8	5	2	5
No advice (%)	4	16	12	35
Perceived mean effectiveness of advice (7 pt scale)*	4	3	4	2
Feel comfortable in giving advice (7 pt scale)*	6	4	5	4
Have available information on quitting/health effects in office (%)	27	26	12	17

*1 = low, 7 = high

TABLE 3—Major Obstacles to Treating Tobacco Use*

	Independent Practice		HMO	
	Dentist	Hygienist	Dentist	Hygienist
Obstacles	%	%	%	%
I am pessimistic about people's ability to change	64	46	70	36
Patients resist referral to cessation clinics	56	52	56	61
I believe or patients believe intervention is not appropriate since tobacco use is a matter of personal choice	49	42	49	36
I have too little time	38	31	42	34
I need further training	37	55	33	52
There is lack of coordination between dentistry and tobacco cessation services	30	31	42	39
I have little confidence in effectiveness of available services	26	11	26	16
There are too few services to which patients can be referred	16	22	7	11

*Respondents asked to choose four of the eight statements listed.

ested in providing cessation information and/or advice. Unfortunately, dental professionals receive little training and have few materials available that would facilitate such actions. Dentists appear more comfortable giving advice to smokeless tobacco users than to smokers. The relevance of chew and snuff use to oral health is the most parsimonious explanation for this finding. The difference between smokeless tobacco use rates in the IP and HMO patient populations may be due to demographic differences, sampling differences, or personnel differences.

The patients surveyed reported receptiveness to cessation advice and, in fact, expect it from a dental professional. Direct feedback from a dentist or hygienist on oral health status, such as visually pointing to tobacco-associated oral lesions, gingival recession, or tooth staining, can produce a "teachable moment" wherein patients are receptive to cessation advice. This receptivity to advice from dentists and hygienists is supported by data from an in-depth interview study of smokeless tobacco users.[6]

Studies of physicians' advice to smoking patients have demonstrated modest but significant efficacy.[7–9] Recent reports indicate that dentists' direct advice also can have a significant effect.[11–14] Similar effects seem likely for smokeless tobacco. The obstacles to implementation include the need for specific cessation training for dentists and hygienists and self-help cessation materials for patients. Specific materials are now becoming available that will assist oral health professionals in getting more directly involved in advising patients who seek their help in order to quit the use of tobacco products.[14]

ACKNOWLEDGMENTS

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REFERENCES

1. US Surgeon General: Surgeon General's Report on Smokeless Tobacco. Washington, DC: Govt Printing Office, 1986.

2. Rouse BA: Epidemiology of smokeless tobacco use: A national study. NCI Monograph, No. 8. Bethesda, MD: National Cancer Institute, 1989.
3. Connolly GN, Winn DM, Hecht SS, Henningfield SE, Walker B, Hoffman D: The reemergence of smokeless tobacco. N Engl J Med 1986; 314:1020–1027.
4. Geboy MJ: Dentists' involvement in smoking cessation counseling: A review and analysis. J Am Dent Assoc 1989; 118:79–83.
5. Gerbert B, Coates T, Zahnd E, Richard RJ, Cummings SR: Dentists as smoking cessation counselors. J Am Dent Assoc 1989; 118:29–32.
6. Severson HH, Eakin EG, Lichtenstein E, Stevens VJ: The inside scoop on the stuff called snuff: An interview study of 94 adult male smokeless tobacco users. J Subst Abuse 1990; 2:77–85.
7. Pederson LL: Compliance with physician advice to quit smoking: A review of the literature. Prev Med 1982; 11:71–84.
8. Thompson RS, Michnich ME, Friedlander L, Gilson B, Grothaus LC, Storer B: Effectiveness of smoking cessation interventions integrated into primary care practice. Med Care 1988; 26:62–76.
9. Glynn TJ: Relative effectiveness of physician-initiated smoking cessation programs. Cancer Bull 1988; 40(6):359.
10. Secker-Walker RH, Hill HC, Solomon LJ, Flynn BS: Smoking cessation practices in dental offices. J Public Health Dent 1987; 47:10–15.
11. Secker-Walker RH, Solomon LJ, Haugh LD, Welsh D, Tatro M, Witham L, Hill HC, Mercier SM: Smoking cessation advice delivered by the dental hygienist: A pilot study. Dent Hyg April 1988; 62(4):186–92.
12. Cohen SJ, Christen AG, Katz BP, Drook CA, Davis BJ, Smith DM, Stookey GK: Counseling medical and dental patients about cigarette smoking: The impact of nicotine gum and chart reminders. Am J Public Health 1987; 77:313–316.
13. Christen AG, McDonald LL, Olson BL, *et al*: Efficacy of nicotine chewing gum in facilitating smoking cessation. J Am Dent Assoc 1984; 108:594.
14. Mecklenberg RE, Christen AG, Berger B, Giff HC, Glynn TJ, Lindsay E, Manley M, Severson HH: How to help your patients stop using tobacco: A National Cancer Institute manual for the oral health team. Bethesda, MD: NCI, US Dept of Health & Human Services, 1990.

Evidence for Gonococcal Transmission within a Correctional System

KAREN H. VAN HOEVEN, MD, WALLACE C. ROONEY, JR., MD, AND STEPHEN C. JOSEPH, MD, MPH

Abstract: In a study to examine sexually transmissable disease occurring within a large correctional system where sexual activity is prohibited, 27 male inmates acquired culture-proven gonorrhea from in-jail sexual activity during a three-month period. These results provide evidence to encourage inmate education about the acquired immunodeficiency syndrome (AIDS) and to support condom distribution programs in correctional facilities. (*Am J Public Health* 1990: 80:1505–1506.)

Introduction

Studies reveal that up to 20 percent of male inmates engage in homosexual activity at some time during their imprisonment.[1] Research protocols which explore homosexual activity in jails and prisons generally use a subjective, questionnaire-type approach, asking prisoners whether they have participated in homosexual activity before or during incarceration.[1–7] Objective evidence for sexual activity occurring during imprisonment has been presented only in a few case reports which describe outbreaks of syphilis or gonorrhea occurring in prisoners incarcerated for prolonged periods.[8,9]

Sexual activity places inmates at risk for acquiring sexually transmitted diseases—including gonorrhea and the acquired immunodeficiency syndrome (AIDS). The risk of infection with the human immunodeficiency virus (HIV) among New York City prisoners during homosexual activity with other inmates is even higher than that of the general population, since studies have shown these inmates to have higher rates of HIV infection at admission to prison than the general population.[10]

Sexual activity is prohibited in many correctional systems, including that of New York City. Thus, the provision of condoms to inmates often is deemed unnecessary and objectionable by correction officials, since condom distribution would imply that rules prohibiting sexual activity are not enforced, or are unenforceable.[11] Yet from a public health perspective, condom distribution to inmates is important to enable inmates to protect themselves from sexually transmitted diseases, including AIDS. This study was designed to answer the following question: is there **objective** evidence that sexual activity occurs **regularly** in a large jail system?

Methods

In 1986, the correctional facilities of the New York City Department of Correction had a capacity to house over 12,500 inmates, and admitted more than 73,000 prisoners. The average daily census was almost always 100 percent or more of capacity. Most of the prisoners were detainees, staying for only a short period of time while awaiting trial or bail. Each prisoner received an admission history and physical exmination which included collecting information concerning a history of gonorrhea, dysuria, or urethral discharge and performing a urinalysis. Any newly admitted inmate who became suspect for gonorrheal infection after admission screening was examined, cultured, and treated for gonorrhea if clinically indicated.

For the months of October, November, and December 1986, the names, prisoner identification numbers, and dates of positive culture for all male New York City inmates with culture-proven gonorrhea were collected. From the files of the New York City Department of Correction, the date of admission to the jail was obtained for these prisoners. If an admission date preceded the date of positive culture by more than one week, the inmate's chart was reviewed and pertinent data were collected. These data included: date symptomatic, collection date and source (urethral, anal, and/or throat) of the specimen, and history of previous gonorrheal infections and treatment received, if any.

Address reprint requests to Karen H. van Hoeven, MD, Department of Pathology, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461. At the time of the study she was with the Bureau of Medical Affairs, New York City Department of Health. Dr. Rooney is Medical Director, Prison Health Service, New York City Department of Health; Dr. Joseph was Commissioner, New York City Department of Health from 1986 through 1989. This paper, submitted to the Journal October 23, 1989, was revised and accepted for publication May 18, 1990.

HIV Seroprevalence among Male IVDUs in Houston, Texas

MARK L. WILLIAMS, PHD

Abstract: HIV seroprevalence was 8.4 percent in a sample of 921 heterosexual male intravenous drug users in Houston, Texas who were not in drug treatment at the time of the study. Males who were Black, injected drugs daily, or had a history of syphilis had greater odds of being HIV positive than participants without those characteristics. (*Am J Public Health* 1990; 80:1507–1509.)

Introduction

The association between acquired immunodeficiency syndrome (AIDS) and intravenous drug use is well documented.[1] The primary means by which intravenous drug users (IVDUs) become infected with the human immunodeficiency virus (HIV), the cause of AIDS, are known.[2,3] However, the risk of an IVDU becoming infected with HIV is not uniform across racial/ethnic groups nor geographic locations.[4–6] As well, rates of HIV seroprevalence have been reported to vary among IVDUs due to the drug injected, frequency of injection, use of shooting galleries, injecting with strangers, the number of sexual partners, and a history of sexually transmitted diseases.[7–11]

The purpose of this study was to examine HIV seroprevalence rates among intravenous drug using males recruited from their natural environments in Houston, Texas. The majority of studies reporting HIV seroprevalence rates among IVDUs have been conducted in drug treatment, detoxification, or some other clinical setting.[2] Few studies have reported on the seroprevalence rates among intravenous drug injectors not in treatment.

Methodology

Subjects for this study were recruited from Houston's intravenous drug using population not in drug treatment. Participants were asked to respond to an interviewer administered survey covering demographic characteristic, drug use, and sexual behaviors. IV drug users were recruited from the city's three predominant racial/ethnic groups by trained, indigenous outreach workers. Three neighborhoods with known high incidences of intravenous drug use were targeted for outreach activities using targeted sampling.[12,13] Although targeted sampling is purposive, it is not random.

Study participants were required to be 18 years, have injected an illicit drug at least once during the six months prior to participation, and not have been in drug treatment for 30 days prior to participation. Outreach workers successfully recruited 1,279 men willing to be interviewed. Of that number, 81 percent agreed to have blood drawn for HIV testing. Only those respondents with complete surveys and HIV testing were considered for inclusion in the data set. Twenty-three participants were excluded because of sexual relationships with men during the previous six months. The resulting study sample of 921 males was 65 percent Black, 24 percent White, and 12 percent Hispanic (primarily Mexican American).

All variables used in the analysis, except HIV serostatus, are self-reported. A respondent's racial/ethnic classification was determined by his stated racial/ethnic identity. Drug use variables analyzed were frequency of injection, length of injected drug use, injection in a shooting gallery, sharing needles with a stranger, number of persons with whom needles were shared, and drug(s) injected. Frequency of injection was categorized as less than weekly, weekly, or daily depending on how often a respondent reported injecting during the six months prior to the interview. Drugs injected were categorized according to the reported drug(s) a respondent said he had injected during the six months before the interview. Respondents who said they injected more than one drug could either have injected the drugs alone or as mixtures.

Variables related to sexual behaviors which were examined were a self-reported history of syphilis, gonorrhea, herpes, and chlamydia. The number of sexual partners a respondent reported during the six months prior to participating in the study was examined.

HIV serostatus was determined by ELISA and Western Blot assays. Blood samples which were repeatedly positive on the ELISA test were screened using Western blot analysis. Samples were classified HIV positive if protein bands P24, P31, and EP41 or EP160 were present.

Univariate relationships were estimated using odds ratios and 95% confidence intervals. Significance was confirmed using Fisher's exact test (not shown).[14] To test the independence of significant univariate associations, a model of those relationships was analyzed using maximum likelihood method of logistic regression.[14,15] Coefficients are reported in independent odds ratios.

Results

HIV infection rates varied by race/ethnicity. As shown in Table 1, Black males had a seroprevalence rate of 10 percent. Seven percent of the White males were HIV positive. Black males were 2.80 times as likely to be HIV positive as Hispanic males.

Frequency of injection was found to be related to seropositivity. Less than weekly users had a 4 percent seropositive rate (Table 1). Eight percent of weekly users were HIV positive. Daily users had the highest number of positive test results, 11 percent. Associations between seroprevalence and self-reported history of syphilis, gonorrhea, herpes, and chlamydia were examined. Only a self-reported history of syphilis was significantly related to seroprevalence. Males who reported having had syphilis were 2.34 times as likely to be HIV positive as those who had not.

To test the independent association between race/ethnicity, frequency of injection, a past history of syphilis, and HIV seroprevalence logistic regression analysis was used. As shown in Table 2, these three variables are inde-

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Address reprint requests to Mark L. Williams, PhD, Affiliated Systems Corp., 1200 Post Oak Blvd., Suite 540, Houston, TX 77056-3104. This paper, submitted to the Journal June 26, 1989, was revised and accepted for publication May 16, 1990.

TABLE 1—Seroprevalence Rates for Male IV Drug Users

	Percent Positive/n(%)*	Odds Ratio	95% Confidence Interval (lower/upper)
Race/Ethnicity			
Hispanic	4/107 (4)	—	—
White	14/214 (7)	1.80	.58, 5.62
Black	59/600 (10)	2.80	1.01, 7.90
Frequency of Injection			
<weekly	8/206 (4)	—	—
weekly	25/325 (8)	2.06	.91, 4.67
daily	44/390 (11)	3.15	1.45, 6.82
Length of Injected Drug Use			
0–2 years	2/63 (3)	—	—
3–8 years	26/269 (10)	3.26	.75, 14.13
≥9 years	49/540 (8)	2.77	.66, 11.66
Injection in a Shooting Gallery			
no	22/227 (9)	—	—
yes	55/672 (8)	.92	.55, 1.54
Sharing Needles With a Stranger			
no	16/251 (6)	—	—
yes	61/670 (9)	1.47	.83, 2.60
Number of Persons with Whom Needles Shared			
none	12/139 (9)	—	—
1–2	37/429 (9)	1.00	.51, 1.98
≥3	28/325 (8)	.91	.45, .80
Drug Injected			
heroin	1/32 (2)	—	—
cocaine and heroin	11/198 (6)	1.82	.23, 14.63
cocaine	44/476 (9)	3.16	.42, 23.69
cocaine and amphetamines	21/215 (10)	3.36	.44, 25.85
Syphilis			
no	61/759 (7)	—	—
yes	16/101 (16)	2.34	1.29, 4.24
Gonorrhea			
no	44/562 (8)	—	—
yes	33/359 (9)	1.19	.074, 1.91
Herpes			
no	76/907 (8)	—	—
yes	1/14 (7)	.84	.11, .652
Chlamydia			
no	76/836 (8)	—	—
yes	1/8 (11)	1.38	.17, 11.14
Number of Sexual Partners			
none	9/75 (12)	—	—
one	21/224 (9)	.76	.33, 1.74
2–3	16/220 (7)	.58	.24, 1.36
≥4	31/402 (8)	.61	.28, 1.35

*rounded to nearest whole percent

TABLE 2—Logistic Regression Model

Variables	Odds Ratio	95% Confidence Intervals
Black	1.39	1.05,1.84
Daily Injection	1.40	1.09,1.77
Syphilis	1.42	1.05,1.92

pendently associated with seropositivity. Each of the variables has approximately equal weight.

Discussion

The results of the analysis show that there is an association between race, frequency of injection, and a past history of syphilis. The relationship between frequency of injection and HIV infection did not vary by drug(s) injected. Heroin users injecting daily were just as likely to be HIV positive as cocaine users injecting daily. Although drug injected may have some pharmacological effect on the injecting behaviors of users, how often an IVDU is injecting, whatever the drug, would appear to be of greater importance. The association between a history of syphilis and HIV infection is far from clear. It would seem likely that the variable is indicative of risk of HIV infection due to behaviors associated with the condition.

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REFERENCES

1. Curran JW, Jaffe HW, Hardy AM, Morgan WM, Selik RM, Dondero TJ: Epidemiology of HIV infection and AIDS in the United States. Science 1988; 239:610–616.
2. Hahn A, Onorato IM, Jones TS, Dougherty J: Prevalence of HIV infection among intravenous drug users in the United States. JAMA 1989; 261:2677–2684.
3. Des Jarlais DC, Wish E, Friedman SR, *et al*: Intravenous drug use and the heterosexual transmission of the human immunodeficiency virus. NY State J Med 1987; 283–286.

4. Selik RM, Castro KG, Pappaioanou M: Racial/Ethnic differences in the risk of AIDS in the United States. Am J Public Health 1988; 78:1539–1545.
5. Lange WR, Snyder FR, Lozovsky D, *et al*: Geographic distribution of human immunodeficiency virus markers in parenteral drug abusers. Am J Public Health 1988; 78:443–446.
6. Selik RM, Castro KG, Pappaioanou M, Buehler JW: Birthplace and the risk of AIDS among Hispanics in the United States. Am J Public Health 1989; 79:836–839.
7. Chaisson RE, Bacchetti P, Osmond D, Brodie B, Sande MA, Moss AR: Cocaine use and HIV infection in intravenous drug users in San Francisco. JAMA 1989; 261:561–565.
8. Des Jarlais DC, Friedman SR: HIV infection among persons who inject illicit drugs: Problems and perspectives. J AIDS 1988; 1:267–273.
9. Friedman SR, Des Jarlais DC, Neaigus A, *et al*: AIDS and the new drug injector. Nature 1989; 339:333–334.
10. Newmeyer JA, Feldman HW, Biernacki P, Watters JK: Preventing AIDS contagion among intravenous drug users. Med Anthropol 1989; 10:167–175.
11. Sasse H, Salmaso S, Conti S, *et al*: Risk behaviors for HIV-1 infection in Italian drug users: Report from a multicenter study. J AIDS 1989; 2:486–496.
12. Sudman S, Sisken ME, Cowan CD: Sampling rare and elusive populations. Science 1988; 240:991–996.
13. Watters JK, Biernacki P: Target sampling: Options for the study of hidden populations. Soc Probl 1989; 36:416–430.
14. SAS Institute Inc.: SAS/STAT User's Guide. Cary, NC: SAS Institute Inc, 1988.
15. Aldrich JH, Nelson FD: Linear Probability, Logit, and Probit Models. Newbury Park, CA: Sage Publications, 1989.

HIV Seroconversion in Two Homosexual Men after Receptive Oral Intercourse with Ejaculation: Implications for Counseling Concerning Safe Sexual Practices

ALAN R. LIFSON, MD, MPH, PAUL M. O'MALLEY, NANCY A. HESSOL, MSPH, SUSAN P. BUCHBINDER, MD,
LYN CANNON, AND GEORGE W. RUTHERFORD, MD

Abstract: Seroconversion for HIV antibody occurred in two homosexual men who reported no anal intercourse for ≥ 5 years and multiple episodes of receptive oral intercourse with ejaculation. Neither man reported intravenous drug use or receipt of blood products. The last antibody-negative specimen was also negative by the polymerase chain reaction and p24 antigen assays. All sexually active persons should be clearly counselled that receptive oral intercourse with ejaculation carries a potential risk of HIV transmission. (*Am J Public Health* 1991; 81:1509–1511.)

Introduction

Studies of homosexual and bisexual men have shown that anal intercourse, especially receptive anal intercourse with ejaculation, is the sexual practice associated with the greatest risk of human immunodeficiency virus (HIV) infection.[1-5] Although orogenital contact has not been statistically associated in these studies with HIV infection, many subjects engaged in multiple sexual practices, making a relatively lower risk more difficult to detect statistically.

Although transmission of HIV through receptive oral intercourse has been suggested in a few brief reports,[4,6,7] these reports have not provided extensive detail about the circumstances surrounding seroconversion. For this reason, there continues to be controversy about the safety of oral intercourse.

We report seroconversion for HIV antibody in two homosexual men who denied anal intercourse for ≥ 5 years and who engaged in multiple episodes of receptive oral intercourse with ejaculation.

Methods

The San Francisco City Clinic Cohort consists of 6,705 homosexual and bisexual men originally recruited from 1978 through 1980 for studies of hepatitis B.[8,9] Men enrolled in our prospective studies were recruited from this original cohort and are evaluated with an annual interview, including a sexual history by a trained staff member which asks about their activities in the preceding four months and for the entire period since their last interview. Study participants also receive an examination and medical history by a physician unaware of the subject's antibody status. Sera were tested for HIV antibody with the ELISA (Organon Teknika, Research Triangle, NC) and confirmed by both the Western blot and immunofluorescence assay (IFA).[10] Testing for HIV-DNA by the polymerase chain reaction (PCR) was conducted on DNA extracted from peripheral blood mononuclear cells using two different *gag* primers.[11] Sera were also tested for p24 antigen (Coulter Immunology, Hialeah, FL).

Results

For both men who seroconverted, in the two years preceding seroconversion at least three specimens were negative by the ELISA assay; the last pre-seroconversion specimen was also negative by PCR and p24 antigen assays. The first post-seroconversion specimen was positive for HIV antibody by the ELISA, IFA and Western blot assays. Both men were also HIV-antibody positive on repeat testing of the first post-seroconversion specimen (including a second serum sample drawn at the same time), as well as on at least one follow-up specimen drawn between four and nine months later.

Both men reported no insertive or receptive anal intercourse for at least five years preceding the estimated date of seroconversion; this history was supported by three previous interviews. Both men also denied use of intravenous drugs,

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From the AIDS Office, Department of Public Health, San Francisco. Address reprint requests to Alan R. Lifson, MD, MPH, AIDS Office, Department of Public Health, 25 Van Ness Avenue, Suite 500, San Francisco, CA 94102. Dr. Rutherford is currently with the California State Department of Health Services. This paper, submitted to the Journal March 2, 1990, was revised and accepted for publication July 13, 1990.

7. Rozenbaum W, Gharakhanian S, Cardon B, Duval E, Coulaud JP: HIV transmission by oral sex. Lancet 1988; 1:1395.
8. Hessol NA, Lifson AR, O'Malley PM, Doll LS, Jaffe HW, Rutherford GW: Prevalence, incidence and progression of human immunodeficiency virus infection in homosexual and bisexual men in hepatitis B vaccine trials, 1978-1988. Am J Epidemiol 1989; 130:1167–1175.
9. Jaffe HW, Darrow WW, Echenberg DF, *et al:* The acquired immunodeficiency syndrome in a cohort of homosexual men: A six-year follow-up study. Ann Intern Med 1985; 103:210–214.
10. Gallo D, Diggs JL, Shell GR, *et al:* Comparison of detection of antibody to the acquired immune deficiency syndrome virus by enzyme immunoassay, immunofluorescence, and Western blot methods. J Clin Microbiol 1986; 23:1049–1051.
11. Lifson AR, Stanley M, Pane J, *et al:* Detection of human immunodeficiency virus DNA using the polymerase chain reaction in a well-characterized group of homosexual and bisexual men. J Infect Dis 1990; 161:436–439.
12. Lifson, AR: Do alternate modes for transmission of human immunodeficiency virus exist? A review. JAMA 1988; 259:1353–1356.
13. Fox PC, Wolff A, Yeh CK, Atkinson JC, Baum BJ: Saliva inhibits HIV-1 infectivity. JADA 1988; 116:635–637.
14. Hessol NA, O'Malley P, Lifson A, *et al:* Incidence and prevalence of HIV infection among homosexual and bisexual men, 1978-1988. *In:* Proceedings of the Fifth International Conference on AIDS, June 4-9, 1989, Montreal (Abstract M.A.0.27).
15. Kingston T: Safe(r) sex guidelines border disputes rage on. Coming Up! September 1988; 9(12):6–7,54.
16. Conkin D: Is oral sex safe? Experts react to new studies that classify fellatio as minimal risk. Bay Area Reporter, August 31, 1989; 19(35):5.
17. Dooley J: To suck or not to suck? An in-depth look at what AIDS educators are telling men about the relative risk of oral sex without a condom. Gay Community News, January 29–February 4, 1989; 16(28):7.

The AIDS-Related Experiences and Practices of Primary Care Physicians in Los Angeles: 1984–89

CHARLES E. LEWIS, MD, AND KATHLEEN MONTGOMERY, PHD

Abstract: Telephone interviews of random samples of Los Angeles primary care physicians in 1984, 1986, and 1989 obtained information about their AIDS-related practice experiences, and sexual history taking. Data from mid-1989 reveal almost 74 percent have worked up at least one patient for AIDS or HIV infection in the past six months and 39.5 percent are caring for at least one patient with AIDS or AIDS-related complex. Self-reported use of appropriate sexual history questions has improved substantially over this five-year period. (*Am J Public Health* 1990; 80:1511–1513.)

Introduction

There has been growing speculation, based primarily upon surveys of physicians' attitudes, that a significant proportion of doctors might fail to provide care to patients found to be infected with the human immunodeficiency virus (HIV) and/or who developed acquired immunodeficiency syndrome (AIDS).[1,2]

Over the past six years, we have conducted three telephone surveys of random samples of primary care physicians in full-time office-based practice in Los Angeles, California (January 1984,[3] summer 1985,[4] and summer 1989). Data presented here illustrate changes over this period in physicians' experiences with patients at risk or infected with HIV.

Methods

Stratified random samples of internists, family practitioners, and general practitioners were drawn to reflect the relative proportion of each of these groups practicing in Los Angeles County. The samples were drawn from data tapes provided by the American Medical Association Physician Masterfile, containing listings of all primary care physicians in full-time office-based practice in Los Angeles—a pool of about 5,000 physicians.[5]

Results

Response Rates and Characteristics of the Sample

The response rates for all three surveys have been above 60 percent; the most recent survey resulted in a 77 percent response rate. These response rates are calculated using as the denominator all physicians actually contacted (see Table 1).

Examination of the demographic characteristics of respondents versus nonrespondents during each survey period has revealed no obvious source of bias. The increase in the proportion of women interviewed over the past five years reflects the growing number of women primary care physicians practicing in Los Angeles County; for Los Angeles as

TABLE 1—Response Rates and Characteristics of Respondent Physicians

	1984 (635)[a]	1986 (279)[b]	1989 (463)[a]
% Response Rates	63.1	60.0[c]	77.0
Specialty			
% General Internal Medicine	42.4	44.1	47.7
% General Practice	26.3	17.2	17.3
% Family Practice	31.3	38.7	35.0
% Solo Practice	50.4	54.5	51.2
% Foreign Medical Graduates[d]	17.3	19.9	21.4
% Female	6.8	9.0	19.7

a) Los Angeles County.
b) Los Angeles SMSA.
c) Response rate represents that for the entire state of California (N=1000).
d) Does not include graduates of Canadian or Western European medical schools.

From the Department of Medicine, UCLA School of Medicine (Lewis), and the Graduate School of Management, University of California, Riverside (Montgomery). Address reprint requests to Charles E. Lewis, MD, Division of General Internal Medicine and Health Services Research, B-558 Factor Building, UCLA School of Medicine, 10833 Le Conte Avenue, Los Angeles, CA 90024-1685. This paper, submitted to the Journal January 23, 1990, was revised and accepted for publication April 20, 1990.

a whole, the proportions are: 11 percent in 1984, 14 percent in 1986, and 16 percent in 1989.[5]

AIDS-Related Practice Experiences

The proportion of physicians who, within the six months prior to being interviewed, had worked up or referred a patient for work up of possible HIV infection, including AIDS, has almost tripled, growing from 27 percent to over 73 percent in the five years between 1984 and 1989. Also, the proportion of physicians who have at least one patient in their practice with AIDS has grown from less than 1 percent in 1984 to almost 40 percent in 1989. While 29 percent of primary care physicians surveyed in 1989 had between one and five AIDS patients in their practice, a small group of these physicians (3 percent) were caring for 15 or more patients with AIDS (see Table 2).

Sexual History Taking Questions

Physicians were asked if they took sexual histories on all their new patients. Those responding affirmatively were asked, "What do these questions cover?"

As Table 2 indicates, the proportion of physicians who volunteered they ask about the sexual orientation of their patients has doubled during this five-year interval, with close to half the physicians in 1989 saying they asked for such information. The proportion reporting they ask patients about the number of sexual partners has tripled in the five-year period.

Physicians' Attitudes

Physicians were also asked the extent to which they think the average physician is uncomfortable discussing sexual matters with homosexual patients. In an attempt to avoid only socially acceptable responses, the question was phrased in general terms, rather than asking about the doctor's own level of discomfort. Table 2 shows that these figures have changed little over the past five years, with nearly three-fourths estimating that their colleagues have moderate or considerable discomfort. The estimated discomfort of others is unrelated to physicians' own reported AIDS-related experiences.

Discussion

The data from these three surveys suggest that, as a group, a significant number of primary care physicians in Los Angeles have responded to the growth in the number of AIDS cases by accepting responsibility for the care of patients with AIDS or AIDS-related complex. Unfortunately, these data do not allow us to estimate the extent to which the patients under care of these physicians represent their established patients who have developed AIDS, or whether these are new patients in the practice. Nor can we distinguish among the 60 percent of physicians who have no AIDS patients in their practice how many have refused to care for a patient with AIDS, as contrasted to those who merely have not yet encountered any patients with AIDS.

It may be reassuring to those responsible for funding physicians' education about AIDS that physicians' awareness of the content of a sexual history has improved substantially during the past five years.

While the data on physicians' sexual history taking is self-reported, there is evidence that their awareness of some key questions that should be asked has increased. Knowledge of these is a prerequisite to their use, but we have no data from patients to validate that these were asked. Despite this improvement, a majority of primary care physicians apparently are not asking questions essential in assessing sexual risks.

The persistent belief over time among physicians that their colleagues are uncomfortable in dealing with homosexual patients remains puzzling, given the increases in contact with AIDS patients and reports of more comprehensive sexual histories. Lacking additional data, we were unable to determine whether physicians' perceptions of colleagues' discomfort is unrelated to their own feelings, or that feelings of discomfort simply are not strong predictors of the willing-

TABLE 2—AIDS-Related Experiences and Practices

	1984[a] (95% CI)	1986[b] (95% CI)	1989[a] (95% CI)
Practice Experience			
Worked up a patient for AIDS or HIV infection in past 6 months	27.0 (23.5, 30.4)	43.0 (37.2, 48.8)	74.7 (70.7, 78.7)
Caring for at least one patient with AIDS or ARC	0.6 (−.002, 1.2)	6.1 (3.3, 8.9)	40.0 (35.5, 44.5)
Sexual History Taking[c]			
Ask patients about their sexual orientation (sex of partners)	23.0 (19.7, 26.3)	20.1 (15.4, 24.8)	42.3 (37.8, 46.8)
Ask patients about number of sexual partners	9.9 (7.6, 12.2)	9.0 (5.6, 12.4)	29.8 (25.6, 34.0)
Ask patients about sexual practices	12.1 (9.6, 14.6)	16.5 (12.1, 20.8)	18.4 (14.9, 21.9)
Physicians' Attitudes			
To what extent do you think the average physician is uncomfortable discussing sexual matters with gay patients?			
Considerable	34.9 (31.2, 38.6)	28.5 (23.0, 34.0)	33.3 (28.8, 37.8)
Moderate	41.2 (37.4, 45.0)	43.7 (37.7, 49.7)	38.6 (33.9, 43.2)
Small to None	23.9 (20.6, 27.2)	27.7 (22.3, 33.1)	28.1 (23.8, 32.4)

a) Los Angeles County: 1984 (N = 635); 1989 (N = 463).
b) Los Angeles SMSA: 1986 (N = 279).
c) Percentages are based on the total number of physicians surveyed.

ness of some physicians to provide care in the face of this extraordinary epidemic.

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REFERENCES

1. Nielsen BP, Gallegos DA, Harris DH: AIDS awareness among rural Utah physicians. Clin Res 1988; 36, Abstract 102.
2. Richardson JL, Lochner T, McGuigan K, Levine AM: Physician attitudes and experiences regarding the care of patients with acquired immunodeficiency syndrome (AIDS) and related disorders (ARC). Med Care 1987; 25:675–685.
3. Lewis CE, Freeman HE, Kaplan SH, Corey CR: The impact of a program to enhance the competencies of primary care physicians in caring for patients with AIDS. J Gen Intern Med 1986; 1:287–294.
4. Lewis CE, Freeman HE, Corey CR: AIDS-related competence of California's primary care physicians. Am J Public Health 1987; 77: 795–799.
5. American Medical Association: AMA Physician Masterfile. Chicago: AMA 1984, 1986, 1989.

Behavioral Change in Longitudinal Studies: Adoption of Condom Use by Homosexual/Bisexual Men

JILL G. JOSEPH, PHD, S. MAURICE ADIB, MD, MPH, JAMES S. KOOPMAN, MD, AND DAVID G. OSTROW, MD, PHD

Abstract: We compared reporting serial cross-sectional prevalence of sexual behavior over time, to reporting individual patterns of behavioral change in a cohort of homosexual men at a six-month interval. Aggregate prevalence rates underestimated the magnitude of change to safer practices, and failed to provide information on relapse to less safe practices. We conclude that it is important to report data based on individual fluctuations in behavior for the evaluation of change over time. (*Am J Public Health* 1990; 80:1513–1514.)

Introduction

During the past few years, the issue of condom use by homosexual/bisexual men in response to the threat of acquired immunodeficiency syndrome (AIDS) has been addressed in several reports from diverse geographic areas. While some reported cross-sectional findings,[1] others have dealt with the more complex task of describing longitudinal behavior patterns.[2] A recent letter by Kelly and St. Lawrence[3] has highlighted the difficulty of documenting relapse to unsafe sex practices through serial cross-sectional studies that mainly monitor relative increases (or decreases) in sexual behavior prevalence.

We have been surveying adoption and maintenance of safer sexual practices, most recently with regard to condom use in receptive anal sex, in a cohort of homosexual/bisexual men in Chicago. This brief report utilizes some findings from that cohort to highlight methodological issues related to reporting behavioral change in longitudinal studies.

Methods

Participants in the Chicago Multicenter AIDS Cohort Study are concurrently enrolled in a longitudinal behavioral and psychosocial investigation. Detailed methods of recruitment and assessment have been described previously.[4,5] Self-administered questionnaires are completed semi-annually after each visit to the study center. Men have already participated in nine such visits and data collecting is ongoing. At visit 4 (V4) in February 1986, 729 men completed a new section of the questionnaire dealing with condom use in receptive anal sex. At visit 5 (V5), complete data were available for 642 men. A total of 601 men completed this section at both visits. Dropout effect was negligible, as participants at V4 and/or V5 did not differ significantly with regard to baseline sociodemographic or behavioral variables. Men in the V4-V5 cohort were predominantly White (89 percent), had a mean age in 1984 of 34.5 years, a mean educational attainment of 16.4 years, and a mean annual income in 1984–85 of $26,300. Forty percent of them were HIV seropositive by V5.

Participants described consistency of condom use during receptive anal sex (always, sometimes, never) with various types of sexual partners, ranging from those with whom they had a steady relationship to anonymous contacts. Two methods for analysis of these data are considered below. The first one defines four levels of condom use in receptive anal sex, from most to least safe.

1. no receptive anal sex;
2. consistent condom use with all partners;
3. inconsistent condom use;
4. no condom use.

This method was applied in serial cross-sectional analyses at V4 and V5. The second method includes these same four components considered in terms of behavioral consistency between V4 and V5. For men in each of the above categories at V4, subsequent behavior at V5 was described in one of three ways:

1. maintenance of the same behavior;
2. change to any less safe practice;
3. change to any safer practice.

Results

Table 1 reports aggregate serial cross-sectional findings from V4 and V5. It shows a slight increase in the prevalence of safer sexual practices (no receptive anal sex or consistent condom use) with some decrease in no condom use. Overall, change reported as aggregate data suggests that prevalence of

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From the Department of Epidemiology, School of Public Health, University of Michigan. Address reprint requests to Jill G. Joseph, PhD, Associate Professor, Department of Epidemiology, School of Public Health, University of Michigan, 109 Observatory, Ann Arbor, MI 48109. This paper, submitted to the Journal March 29, 1989, was revised and accepted for publication April 22, 1990.

TABLE 1—Serial Cross-Sectional Prevalence of Condom Use in Receptive Anal Sex at Visit 4 (V4) and Visit 5 (V5)

	V4 N (%)	V5 N (%)
No receptive anal sex	375 (52)	341 (53)
Consistent condom use	66 (9)	78 (12)
Inconsistent condom use	96 (13)	94 (15)
Condoms never used	192 (26)	129 (20)
Total	729	642

safer sexual practices increased somewhat over a six-month interval.

A second approach to assess risk reduction takes account of intra-individual stability or instability in behavior and is shown in Table 2. Prevalence of intra-individual change in the V4-V5 cohort was 41 percent, much higher than suggested in Table 1. Among those maintaining the same behavior, a majority maintained safer practices, but 49 percent of those never using condoms maintained such a practice over six months. Intra-individual change to safer sexual practices was reported in 24 percent of the cohort, but this approach revealed relapse to less safe sex in 17 percent of participants.

TABLE 2—Individual Patterns of Maintenance and Change in Safer Sexual Practices in Receptive Anal Sex Over a Six-month Period

Practices at V4	Base at V4 N (%)	Maintained at V5 N (%)	Changed to less safe N (%)	Changed to safer N (%)
No receptive anal sex	307 (51)	239 (78)	68 (22)	— —*
Consistent condom use	56 (9)	24 (43)	13 (23)	19 (34)
Inconsistent condom use	85 (14)	27 (32)	23 (27)	35 (41)
Condoms never used	153 (26)	62 (49)	— —*	91 (59)
Total (N = 601)	601	352 (59)	104 (17)	145 (24)

*Such change was not possible at V5, as participants were already at most extreme categories at V4.

Discussion

Intra-individual fluctuations in individual risk-relevant behaviors have been reported in many longitudinal studies of homosexual men.[6] As early as 1985, a report from San Francisco noted that over the course of one year, approximately half the men observed in one cohort had changed their behavior, from completely or probably safe to probably or definitely risky.[7] Aggregate measures, as we demonstrated, can underestimate the magnitude of change to safer sexual practices, but even more important, they often fail to convey important information on adoption, maintenance and relapse in individual patterns of behavior over time. They are therefore, an essential part of evaluating long-term behavioral change, whether the change is related to HIV/AIDS or to other conditions.

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REFERENCES

1. Valdiserri RO, Lyter D, Leviton LC, Callahan CM, Kingsley LA, Rinaldo CR: Variables influencing condom use in a cohort of gay and bisexual men. Am J Public Health 1988; 78:801–805.
2. Schechter MT, Craib KJP, Willoughby B, *et al:* Patterns of sexual behavior and condom use in a cohort of homosexual men. Am J Public Health 1988; 78:1535–1539.
3. Kelly JA, St. Lawrence JS: Risk behavior change in gay men. Am J Public Health 1990; 80:351.
4. Joseph JG, Montgomery SB, Emmons CA, *et al:* Magnitude and determinants of behavioral risk reduction: Longitudinal analysis of a cohort at risk for AIDS. Psychol Health 1987; 1:73–96.
5. Joseph JG, Montgomery SB, Emmons CA, *et al:* Perceived risk of AIDS: Assessing the behavioral and psychological consequences in a cohort of gay men. J Appl Soc Psychol 1987; 17:231–250.
6. Becker MH, Joseph JG: AIDS and behavioral change to reduce risk: A review. Am J Public Health 1988; 78:394–410.
7. Stall R, McKusick L, Wiley JA, Coates TJ, Ostrow DG: Alcohol and drug use during sexual activity and compliance with safe sex guidelines for AIDS: The AIDS Behavioral Research Project. Health Educ 1986; 13:359–371.

Seat Belt Use in Cars with Air Bags

ALLAN F. WILLIAMS, PHD, JOANN K. WELLS, BS, AND ADRIAN K. LUND, PHD

Abstract: Seat belt use was observed in 1,628 cars with air bags and manual belts and 34,223 cars with manual seat belts only. Sixty-six percent of drivers in cars with air bags wore seat belts compared to 63 percent of drivers in cars with manual belts only. The study found no evidence for the speculation that drivers with air bags will reduce their seat belt use because they believe an air bag alone provides sufficient protection. (*Am J Public Health* 1990; 80:1514–1516.)

Introduction

Federal regulations in the United States require that all passenger cars manufactured in 1990 and after be equipped with automatic restraints. During the phase-in period for this requirement, manufacturers had to equip 10 percent of their passenger cars with automatic restraints in the 1987 model year, 25 percent in 1988, and 40 percent in 1989. Two types of restraint systems have been used to meet the requirement: air bags that inflate automatically in frontal crashes, and seat belts that automatically fasten around occupants when they enter or start the car. When the automatic restraint requirement was being phased in, most manufacturers met it by installing automatic seat belts rather than air bags. Increasingly, air bags have been offered. The percentage of automatic restraint cars sold with air bags was about 10 percent in the 1987 model year and is projected to be 30 percent in

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From the Insurance Institute for Highway Safety. Address reprint requests to Allan F. Williams, PhD, Insurance Institute for Highway Safety, 1005 N. Glebe Road, Arlington, VA 22201. This paper, submitted to the Journal April 23, 1990, was revised and accepted for publication June 29, 1990.

1990 models.[1] Based on announced plans of manufacturers, air bags will be the predominant automatic restraint offered in the 1990s.

Cars with driver side air bags (as well as those few that currently have right front passenger side air bags) are also equipped with manual lap and shoulder belts to meet federal requirements for lateral and rollover crash protection. Together, air bags and lap/shoulder belts provide the most effective restraint protection available. There has been concern that occupants might be less likely to use a manual lap/shoulder belt in vehicles with air bags than in cars that had *only* manual lap/shoulder belts, because they may erroneously assume that the air bag alone provides full or sufficient crash protection. To the extent that this occurred, it would lessen the crash protection otherwise achievable through the combination of air bags and seat belts. To provide information on this question, a survey of belt use in late model cars with and without air bags was undertaken.

Methods

Seat belt use by drivers and right front seat passengers was observed in four metropolitan areas in the fall of 1989. The areas surveyed were metropolitan Chicago, Los Angeles, Philadelphia, and the Maryland and Virginia suburbs of Washington, DC. In each of these areas, seat belt use is required by state law.

To provide a sample of late model cars, observations were restricted to cars with high center mounted brake lights. These lights have been required on all new cars beginning with the 1986 model year. (Some 1985 cars also had high center mounted brake lights, but these were subsequently eliminated from the study sample.)

In each metropolitan area, observations were made at 64 sites, typically intersections with traffic lights. The sites were distributed throughout the metropolitan regions but were concentrated in affluent areas to maximize the likelihood of observing late model cars. Observations at a site were made during either morning (7:00–9:30 am) or evening (4:00–6:30 pm) rush hours. Sites and observation procedures were the same as those used in a previous survey.[2]

In addition to observing shoulder belt use, observers recorded license plate numbers of the vehicles, which were used to obtain vehicle identification numbers (VINs) from motor vehicle departments in Illinois, California, Pennsylvania, Maryland, and Virginia. VINs provide information on car model, model year, and type of restraint system. Using this technique, errors in identifying the vehicle observed are possible only in rare instances, such as the license number was recorded incorrectly or the owner had transferred the license plate from another car to the one observed and the department of motor vehicles had not yet updated their files.

Results

Seat belt use was observed in 45,466 1986–90 model year cars; complete VIN information was available for 97 percent of these vehicles. Of these 44,102 cars, 1,628 were equipped with air bags and lap/shoulder manual belts; 34,223 were equipped with manual lap/shoulder belt systems only. Driver belt use rates were about the same in cars with and without air bags (Table 1). Overall, 66 percent of drivers in cars with air bags used seat belts compared to 63 percent of drivers in cars with manual belts only. Belt use rates of drivers with and without air bags were about the same in each metropolitan area, although belt use rates were substantially higher in Los Angeles and the Washington, DC area than in Chicago and Philadelphia.

TABLE 1—Driver Manual Belt Use in Cars with Air Bags and in Cars with Manual Belts Only, by Metropolitan Area

Metropolitan Area	Percent (N) Using Belts	
	Air Bag Cars	Manual Belt Cars
Chicago	51 (355)	53 (10441)
Los Angeles	73 (684)	71 (8540)
Philadelphia	59 (317)	57 (8521)
Washington, DC	75 (272)	76 (6721)
Total	66 (1628)	63 (34223)

Only a few of the observed cars had a right front passenger air bag and a right front passenger present (N = 22); 50 percent of these passengers were belted. For right front passengers in cars with driver side air bags only, belt use was 56 percent (N = 235) compared to 54 percent (N = 5473) in cars without air bags. Belt use rates for drivers in cars that had right front passengers present was 64 percent in cars with driver side air bags only and 59 percent in cars without air bags.

Table 2 presents driver belt use in cars with and without air bags by manufacturer. Driver belt use was as high or higher in air bag equipped cars as in cars without air bags except for the Chrysler models, which had a slightly lower use rate.

Table 3 presents driver belt use in cars with and without air bags for specific models of cars that were observed in sufficient numbers to make such comparisons. These data indicate that belt use was usually about the same or slightly higher in cars with air bags.

Cars with air bags in the study sample were on average newer than cars with manual belts only. Table 4 presents data on cars with and without air bags by model year. Belt use increased slightly by model year both for cars with and without air bags. For each model year, driver belt use was as high or higher in air bag cars as in cars without air bags.

Discussion

There is no evidence from this survey that the presence of an air bag lessens the likelihood of using a seat belt. Manual seat belt use by drivers of cars with driver side air bags was about the same as for drivers of late model cars that did not have air bags. In cars with drivers and right front passengers

TABLE 2—Driver Manual Belt Use in Cars with Air Bags and in Cars with Manual Belts Only, by Manufacturer

Manufacturer*	Percent (N) Using Belts	
	Air Bag Cars	Manual Belt Cars
BMW	75 (101)	62 (587)
Chrysler	54 (243)	59 (2852)
Ford	64 (157)	59 (5624)
Honda	73 (166)	73 (4186)
Mercedes	64 (772)	—
Volvo	81 (142)	78 (624)
Total	66	64
Total (excluding Mercedes)	67	64

*Forty-seven air bag cars of low volume manufacturers (driver belt use 68 percent) not included.

TABLE 3—Driver Manual Belt Use in Cars with Air Bags and in Cars with Manual Belts Only, by Make/Series

Make/Series	Percent (N) Using Belts	
	Air Bag Cars	Manual Belt Cars
BMW 5,6,7 Series; M6	76 (96)	75 (88)
Chrysler LeBaron Sedan, Convertible	53 (103)	50 (142)
Chrysler LeBaron Coupe	60 (57)	48 (31)
Dodge Daytona	47 (66)	41 (39)
Ford Tempo	57 (46)	59 (282)
Lincoln Continental	68 (90)	66 (77)
Acura Legend	73 (166)	71 (348)
Volvo 740, 760	82 (133)	81 (325)

TABLE 4—Driver Manual Belt Use in Cars with Air Bags and in Cars with Manual Belts Only, by Model Year

Model Year	Percent (N) Using Belts	
	Air Bag Cars	Manual Belt Cars
1986	63 (185)	63 (9781)
1987	65 (319)	63 (9696)
1988	67 (434)	62 (8673)
1989	66 (630)	64 (5994)
1990	67 (60)	66 (79)

present, driver belt use was eight percentage points higher than passenger belt use in cars with driver side air bags and five percentage points higher in cars with manual belts only. If air bags reduced belt use, driver belt use in these air bag equipped cars would have been much lower relative to passenger use.

Manual belt use rates were higher than average in this survey because the observations were restricted to late model cars and were concentrated in affluent areas in states with laws requiring belt use. All of these factors elevate seat belt use.[3,4] Most of the nation's population (89 percent) is now covered by belt use laws; belt use rates in cars with and without air bags would be expected to be lower in the 14 remaining states without laws. Nevertheless, many drivers and right front passengers observed were not using seat belts even though required by law to do so. An important goal is to increase seat belt use both in cars with and without air bags.

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REFERENCES

1. US Dept of Transportation: Evaluation Plan; Federal Motor Vehicle Safety Standards; Passenger Car Front Seat Occupant Protection. Federal Register 1990; 55:1586–1591.
2. Williams AF, Wells JD, Lund AK, Teed N: Observed use of automatic seat belts in 1987 cars. Accident Anal Prev 1989; 21:427–433.
3. National Highway Traffic Safety Administration: Restraint System Usage in the Traffic Population, 1988 Annual Report. (DOT HS 807 447). Washington, DC: US Department of Transportation, 1989.
4. Lund AK: Voluntary seat belt use among US drivers: Geographic, socioeconomic and demographic variation. Accident Anal Prev 1986; 18:43–50.

Age, Sex, and Road-Use Patterns of Motor Vehicular Trauma in Rhode Island: A Population-Based Hospital Emergency Department Study

IAN R.H. ROCKETT, PHD, MPH, ELLICE S. LIEBERMAN, MD, DRPH, WILLIAM H. HOLLINSHEAD, MD, MPH,
SANDRA L. PUTNAM, PHD, AND HENRY C. THODE, PHD

Abstract: Population-based hospital emergency department data on motor vehicle traffic trauma in Rhode Island, 1984–85, are analyzed by age, sex, and road-use status. Annualized rates of overall and severe trauma were 1,195 cases (95% confidence interval [CI] = 1,164, 1,225) and 102 cases (95% CI = 94, 111) per 100,000 population, respectively. Overall and severe rates peaked at ages 15–24 years. Male rate excesses were most pronounced for motorcycle and pedal cycle trauma. (*Am J Public Health* 1990; 80:1516–1518.)

Introduction

Population-based data on nonfatal motor vehicular trauma are not collected routinely. In population-based studies, case selection has typically been restricted in terms of injury diagnoses, road-use status, and age of population.[1–5] An exception is the Northeastern Ohio Trauma Study, in which a comprehensive sample of motor vehicular trauma cases was drawn from regionwide hospital emergency department records.[6] Adopting a similar methodology, the Rhode Island Department of Health instituted a motor vehicular trauma surveillance system.[7] This report documents patterns of motor vehicle traffic trauma rates for Rhode Island by age, sex, and road-use status.

Methods

Cases represent a 25 percent systematic sample of first encounter motor vehicle traffic trauma patients treated during 1984 and 1985 in the emergency departments of the 12 Rhode Island civilian, acute care, nonpsychiatric hospitals with an emergency facility. They include only Rhode Island patients and fatalities (on RI roads) with known age and sex, who were or could be assigned an ICD-9CM external cause of injury

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From the Department of Health, Leisure, and Safety, and the Health Services Research Center, University of Tennessee, Knoxville; Department of Maternal and Child Health, Harvard School of Public Health, and Department of Obstetrics and Gynecology, Brigham and Women's Hospital, Boston; Division of Family Health, Rhode Island Department of Health; and the Injury Prevention and Analysis Group, Brookhaven National Laboratory. Address reprint requests to Ian R.H. Rockett, PhD, MPH, Department of Health, Leisure, and Safety, University of Tennessee, 1914 Andy Holt Avenue, Knoxville, TN 37996-2700. This paper, submitted to the Journal October 13, 1989, was revised and accepted for publication May 17, 1990.

code (E-code) within ranges E810–E816 and E818–E819. Additional information on the methodology is available elsewhere.[7,8]

Numerators in the overall rates represent annual averages estimated from emergency department cases weighted for the inverse of the sampling fraction. Severe cases were a combination of weighted hospital nonfatal admissions and total fatalities; their initial age groups were collapsed in order to address the small numbers. For both sets of rates, denominators are mid-period age- and sex-stratified population estimates that were based on the 1980 Census for Rhode Island[9] and an official projection of the State population for July 1, 1985.[10]

Results

Overall Trauma

Sample cases numbered 6,268; 92 percent involved Rhode Island residents, and constituted the focus of this report. The estimated 1984–85 average annual number of motor vehicular trauma cases among Rhode Island residents was 11,538, and the corresponding rate was 1,195 cases per 100,000 population (Table 1). Motor vehicle occupants contributed three-fourths of the cases.

The estimated annualized motor vehicular trauma rate for males was 1,311 per 100,000 population, and that for females was 1,080. Irrespective of sex, the rates peaked at ages 15–19 years.* Although varying by sex, this age group also registered the highest rates in the motor vehicle occupant, motorcyclist, and other/unspecified road-use categories. Rates for the pedestrian, pedal cyclist and motorcyclist categories peaked at ages 5–9, 10–14, and 20–24 years, respectively.*

Rates were lowest at the extremes of the age spectrum, and were 21 percent greater for males than females. Male predominance was most evident in the motorcyclist category, where the relative risk exceeded five.

Severe Trauma

The estimated annualized rate of severe trauma (fatalities and hospital admissions) was 103 per 100,000 population (Table 2); 64 percent were in the motor vehicle occupant category; pedestrian and motorcycle trauma cases together comprised almost 30 percent. Respective estimated rates for males and females were 133 and 74 per 100,000 population.

Regardless of sex, the severe trauma rate reached a zenith at ages 15–24 years, and was lowest at ages 0–14 and 45–64. The highest rates in the motor vehicle occupant, motorcyclist and other/unspecified road-use categories were registered in the 15–24 age group. Rates peaked at 0–14 years in the pedal cyclist category. There was a bimodal distribution in the pedestrian category, with rates highest at the extreme ages. Males manifested an 80 percent greater risk of sustaining severe motor vehicular trauma than females. In the motorcyclist category, the overall relative risk for males was nine.

Discussion

Overall trauma rates are probably underestimated here because cases involving individuals who refrained from seeking treatment or were self-treated and those treated in out-of-state hospitals or other health care facilities were outside the study population. Estimated severe trauma rates more closely approximate true rates than do the rates of overall trauma, since they are less likely to be influenced by differential discretionary care-seeking behavior among population sub-groups.

Although the Rhode Island study is more recent than the Ohio study,[6] comparisons are instructive. During the respective time frames of these two studies, neither population was covered by a motorcycle helmet law or by a universal seat belt use law. For every motor vehicle traffic trauma fatality among Rhode Island residents, there were an estimated 11 hospital admissions and 130 patients treated in hospital emergency departments. Respective figures for Northeastern Ohio were one, 11, and 137. Other remarkable similarities are apparent in patterns of overall trauma rates by age, sex, and road-use status as, for example, the excess rates for males and young adults. Severe trauma rates were not computed in the Ohio study.

Experience from the two studies underscores the need for comprehensive E-coding of motor vehicular trauma in hospital records. Also essential in identifying high-risk populations, and designing, targeting, and evaluating appropriate interventions, is compilation and analysis of environmental and behavioral data, such as road configurations at trauma sites and motorist usage of safety devices, alcohol, and other drugs.

Rhode Island was the second state in the nation to enact a child restraint use law. The current data were collected as the founding activity of a nascent injury control unit at the Rhode Island Department of Health with the expectation that they would be useful in advocacy for, and evaluation of, a universal seat belt use law. Unfortunately, Rhode Island remains in the minority of states without such a law. Ironically, absence of a law enabled the Rhode Island

TABLE 1—Number and Percentage of Resident Cases in a 25% Motor Vehicular Trauma Sample by Road-use Status for Persons Treated in Hospital Emergency Departments, and Associated Annualized Rates per 100,000 Population, Rhode Island, 1984–85

Road-use Status	Sample No.	Sample %	Rate	95% Confidence Interval
Motor vehicle occupant	4,319	74.9	894	868, 921
Motorized cyclist	318	5.5	66	59, 73
Pedal cyclist	109	1.9	23	18, 27
Pedestrian	273	4.7	57	50, 63
Other/unspecified	750	13.0	155	144, 166
Total	5,769	100.0	1,195	1,164, 1,225

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*Detailed age-, and sex- and road-use-specific data pertaining to both overall and severe motor vehicular trauma are available from the first author upon request.

TABLE 2—Number and Percentage of Resident Motor Vehicular Trauma Fatalities and Nonfatal Hospital Admissions by Road-use Status, and Associated Annualized Rates per 100,000 Population, Rhode Island, 1984–85

Road-use Status	No.		%‡	Rate	95% Confidence Interval
	Fatalities*	Admissions†			
Motor vehicle occupant	84	295	63.8	65	58, 73
Motorized cyclist	18	61	13.2	14	10, 17
Pedal cyclist	4	10	2.2	2	1, 4
Pedestrian	50	67	16.1	16	13, 20
Other/unspecified	21	18	4.7	5	3, 7
Total	177	451	100.0	103	94, 111

*Fatalities represent a complete enumeration of residents who died due to motor vehicular trauma sustained on in-state public roads in 1984 or 1985. The data source is the Division of Vital Statistics, Rhode Island Department of Health.

†Admissions derive from resident cases in a 25% sample for persons treated in hospital emergency departments for motor vehicular trauma. The 35 fatal emergency department cases are excluded.

‡Percentages are based on a combination of total fatalities and weighted nonfatal admissions. The weights are the inverse of the sampling fraction; four.

population to serve as an external control in an ongoing epidemiologic evaluation of the New York State law.[8,11]

The surveillance system which is described here provided data for an alcohol-related injury intervention study.[12] Rhode Island has now established an Office of Injury Control and implemented a variety of interventions aimed at reducing both vehicular and nonvehicular trauma. E-coding by all hospitals is a top priority of the Office, and geographic study of trauma sites is also planned. Both seat belt and helmet use bills were introduced again in the 1990 session of the State Legislature by the Governor.

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REFERENCES

1. Annegers JF, Grabow JD, Kurland LT, *et al:* The incidence, causes and secular trends of head trauma in Olmstead County, Minnesota, 1953–1974. Neurology 1980; 30:912–919.
2. Whitman S, Coonley-Hoganson R, Desai BT: Comparative head trauma experience in two socioeconomically different Chicago-area communities: A population study. Am J Epidemiol 1984; 119:570–580.
3. Kraus JF, Riggins RS, Franti CE: Some epidemiologic features of motorcycle collision injuries: I. Introduction, methods and factors associated with incidence. Am J Epidemiol 1975; 102:74–98.
4. Kraus JF, Franti CE, Riggins RS, *et al:* Incidence of traumatic spinal cord lesions. J Chronic Dis 1974; 23:471–492.
5. Gallagher SS, Finison K, Guyer B, *et al:* The incidence of injuries among 87,000 Massachusetts children and adolescents: Results of the 1980–81 Statewide Childhood Injury Prevention Program surveillance system. Am J Public Health 1984; 74:1340–1347.
6. Barancik JI, Chatterjee BF, Greene-Cradden YC, *et al:* Motor vehicle trauma in Northeastern Ohio. I: Incidence and outcome by age, sex, and road-use category. Am J Epidemiol 1986; 123:846–861.
7. Rockett I, Hollinshead WH, Lieberman E: A statewide motor vehicle injury surveillance system. RI Med J 1986; 69:67–70.
8. Rockett IRH, Hollinshead WH, Lieberman ES: A preliminary assessment of change in motor vehicle trauma incidence and outcome: Rhode Island, 1984–85. Bull NY Acad Med 1988; 64:750–756.
9. US Department of Commerce: 1980 Census of Population. General Population Characteristics, Rhode Island. Washington, DC: Govt Printing Office, 1981.
10. Statewide Planning Program: Monthly Progress Report for October 1986. Providence, RI: Rhode Island Department of Administration, 1986.
11. Barancik JI, Kramer CF, Thode HC, Harris D: Epidemiology of motor vehicle injuries in Suffolk County, New York, before and after enactment of the New York State seat belt use law: Preliminary assessment of occurrence and severity. Bull NY Acad Med 1988; 64:742–749.
12. Putnam SL: Planning, development and process issues in the Rhode Island Alcohol-Related Injury Prevention Project. *In:* Giesbrecht N, Conley P, Denniston R, *et al* (eds): Research, Action and the Community: Experiences in the Prevention of Alcohol and other Drug Problems. OSAP Prevention Monograph-4. Rockville, MD: Office for Substance Abuse Prevention, DHHS Publication No. (ADM) 89–1651, 1990; 183–195.

Evaluation of the Efficacy and Cost Effectiveness of Health Education Methods to Increase Medication Adherence among Adults with Asthma

Richard A. Windsor, PhD, MPH, William C. Bailey, MD, James M. Richards, Jr., PhD,
Brynda Manzella, MPH, Sen-Jaw Soong, PhD, and Michael Brooks, EdD

Abstract: We randomized 135 adult asthma patients to a control group, and 132 patients to an experimental group which received a special health education intervention. Four adherence measures were documented at baseline and 12-month follow-up: correct inhaler use, inhaler adherence, medication adherence, and total adherence rating. Costs to routinely deliver the intervention were $32.03/patient. Experimental group patients exhibited a significantly higher level of improvement in adherence (44 percent) than control group patients (2 percent). (*Am J Public Health* 1990; 80:1519–1521.)

Introduction

The prevalence of asthma among adults in the United States is 3–6 percent.[1-3] There are over 500,000 hospitalizations and 3,500 deaths from asthma per year.[3] Multiple studies of adults with asthma confirm regimen adherence levels of only 30–40 percent.[4-7] Educational intervention studies have reported methodological problems, used small sample sizes, lacked cost analyses, and presented inconclusive results.[8] This evaluation study was designed to document *efficacy* (the level of behavioral impact produced by a health education program with optimal resources) and *costs* (personnel and materials needed to routinely deliver the program).

Methods

The study was conducted at a university-based comprehensive pulmonary medicine clinic. Patients who had a primary diagnosis of asthma, used medications daily, and met the following diagnostic criteria were eligible: recurrent episodes of wheezing or dyspnea; objective evidence of significantly increased airway resistance during episodes; and improvement in the airway when symptom-free.[9-10] Of the 280 adults ≥ 17 years meeting the criteria screened between April 1986 to March 1987, 267 (95 percent) participated.

Following informed consent and baseline assessment, 135 patients were randomized to a control group, and 132 patients to an experimental group. Prior to randomization, patients were stratified by level of asthma severity within each of 11 physician practices (see Appendix). Using pilot study data, an estimated possible improvement in adherence of 20 percent or more for 12 months,[4-8,11-15] and an anticipated 10 percent attrition rate, we defined a need for at least 120 patients in each study group.[16,17]

Health Education Intervention

The experimental group received a peak flow meter and a standardized program from a health education specialist:

- A 30-minute one-to-one session with instruction on peak flow meter use, inhaler use skills, and use of *A Self-Help Guide to Asthma Control;*[18]
- A 60-minute asthma support group session of four to six patients and asthma control partners, and
- Two brief telephone reinforcement calls within one month of the group session.

One-to-one discussions and a review of all methods and materials were conducted with clinic nurses and all 11 participating pulmonary physicians. Four group sessions with patients were held to develop the intervention. All educational components and evaluation methods were pilot tested.

Measurement

All patients received a baseline and 12-month follow-up medical and behavioral assessment including four behavioral outcomes: 1) correct inhaler use, 2) inhaler adherence, 3) medication adherence, and 4) total adherence rating. A 10-item observational checklist documented inhaler use (IU) and skill.[19] Medication adherence (MA) and inhaler adherence (IA) were assessed using instruments adapted from the literature.[20-22] Psychometric analyses (Cronbach Alpha) confirmed adequate reliability: r (MA) = .69, r (IA) = .64, and r (IU) = .78. Items analyses confirmed minimum item discrimination coefficients: r ≥ .20. A Total Adherence Score was derived by combining patient scores on the medication and inhaler adherence scales.

Over 85 percent of the patients used an inhaled bronchodilator and continuous theophylline. Theophylline levels were analyzed on the two follow-up assessment days to indirectly corroborate patient adherence reports. Patients were informed that their theophylline level would be analyzed to assess adherence. While theophylline level is an imperfect measure of adherence and does not reflect adherence to non-theophylline medications, behavioral studies of adults who know a method is available to validate self-reports document a significant reduction in deception rates (bogus pipeline or pipeline effect).[23,24]

Behavioral Intervention Results

A comparison of the 13 patients who refused (5 percent refusal rate) versus the 267 who participated confirmed no baseline differences by gender, race, age, education, and asthma severity. Data in Table 1 confirm group equivalence at baseline. Thirty-four of the control group patients (25 percent) and eight of the experimental group patients (6 percent) were lost to follow-up. Analyses of baseline data confirmed that the 42 dropouts were not significantly different from the 238 participants. Within the intervention group, use of the *Asthma Guide*[18] was reported by 124 patients (94 percent); 110 (89 percent) participated in the group session

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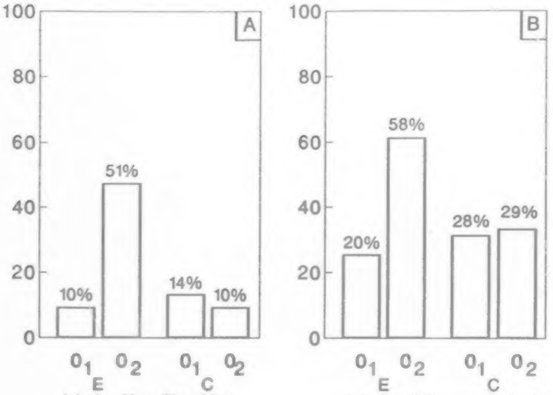
From the Department of Health Behavior, University of Alabama at Birmingham, School of Public Health (Windsor), and the Lung Health Center, UAB School of Medicine, and VA Medical Center (Bailey, Richards, Manzella, Soong, Brooks). Address reprint requests to Richard A. Windsor, PhD, MPH, Chair, Department of Health Behavior, UAT School of Public Health, UAB Station—CBB 502, Birmingham, AL 35294. This paper, submitted to the Journal June 26, 1989, was revised and accepted for publication May 1, 1990.

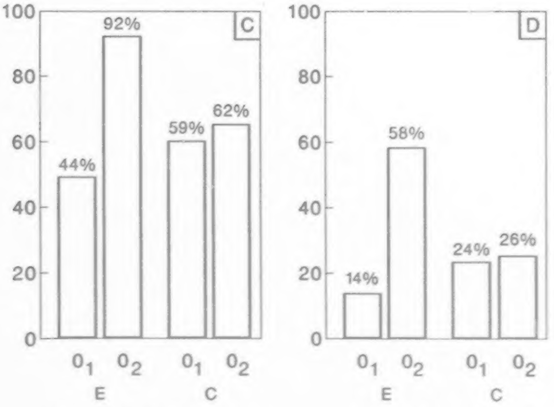
TABLE 1—Base-Line Patient Characteristics by Study Group

Characteristics	Control Group N = 135	Experimental Group N = 132
% Female	71	6[illegible]
% Black	28	32
Median Age (years)	49	50
Median Years of Education	13	13
% Current Smoker	13	10
% Asthma Severity		
Mild	39	37
Moderate	45	48
Severe	17	16



Inhaler Use Checklist All Ten Items - Correct

Inhaler Adherence Scale All Six Items - Yes



Medication Adherence Scale All Six Items - Yes

Total Adherence Score All Twelve Items - Yes

FIGURE 1—Behavioral Impact by Type and Study Group

and 124 patients (94 percent) received both reinforcement calls.

The behavioral impact of the intervention is presented in Figure 1. Using a 95% confidence interval (CI) to evaluate differences between rates of group improvement, a consistent pattern of adherence was confirmed for a 12-month period. Significant improvements for the intervention group in inhaler skills use (CI = .29, .61), inhaler adherence (CI = .24, .50), medication adherence (CI = .31, .57), and total adherence score 100 percent adherence (CI = .28, .56) were observed. Little behavior change was observed in the control group.

Cost Estimation and Analysis

Patient time and intervention development costs were not used to compute program costs. While an MPH-health education specialist provided the intervention, a nurse would be the typical provider. We used a salary of $25,000 plus a fringe benefit rate of 20 percent to estimate nursing cost: $30,000 per year/2080 hr = $14.42/hr. Total personnel time costs were $24.30: Component #1 = $7.21, Component #2 = $14.42 and Component #3 = $2.40. The *Asthma Guide*[18] cost $8.00 for a Total Intervention Cost = $32.03/patient. The total control group costs for a 10-minute general discussion about the importance of adherence and two brief follow-up contacts for a total of 15 minutes were $3.61/patient.[25,26] Cost analyses presented in Table 2 confirmed the need to allocate resources to significantly increase patient adherence.

Discussion

The increases in adherence observed in our intervention group exceeded those of previous reports. While some proportion of patient self-reports of adherence may represent a "social desirability" response, psychometric analyses indicated good measurement of behavioral impact. Our high recruitment rate confirmed that adults with asthma are interested in taking an active role in their self-management and will participate in a health education program integrated into routine asthma care. Near unanimous positive anecdotal comments about the intervention by nurses, physicians, and patients were observed. Feasibility, patient and provider acceptance was very high. From an administrative and programmatic perspective, our experience indicated that this type of intervention has potential for adaptation by other asthma programs. However, in our attempt to maximize efficacy, an intervention was provided which may be too resource intensive and costly for other asthma care sites.

While the methods and results reported here and elsewhere were encouraging,[8,18,27] future health education research needs to evaluate the impact of a streamlined health education program on health services utilization, e.g., am-

TABLE 2—Cost Effectiveness of the Health Education Program

Groups	Total Cost per Patient	Total Adherence Score Improvement	Cost Effectiveness*
Control Group (N = 135)	$ 3.61	+2%	$243.68
Experimental Group (N = 132)	$32.03	+44%	$ 96.09

*Cost Effectiveness = Total cost per group ÷ Adherence score increase

bulatory care, emergency care and hospitalization, and clinical outcomes.

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The findings were presented at the 117th annual meeting, American Public Health Association, October 25, 1989, Chicago, Illinois.

APPENDIX

Dear Doctor,

As part of the Asthma Education Project, the severity of your patient's asthma based on their current regimen is needed.

Please circle the appropriate severity level:

0—Mild—generally wheeze-free, responds well to medication.
1—Moderate—occasional wheezing with periods of significant wheezing.
2—Severe—constant wheezing, does not respond well to medications, steroid dependent

Please encourage your patient to participate in the Asthma Education Project.

Thank you,

The Asthma Project Staff

REFERENCES

1. Broader I, Higgins MW, Mathews KP, *et al:* Epidemiology of asthma and allergic rhinitis in a total community, Tecumseh, Michigan. III. Second survey of the community. J Allergy Clin Immunol 1974; 53:127–138.
2. National Institute of Allergy and Infectious Diseases: Asthma and Other Allergic Disease: Report of the NIAID Task Force. Washington, DC: National Institutes of Health, 1979.
3. Evans R, Mullally D, Wilson RW, *et al:* National trends in the morbidity and mortality of asthma in the USA: Prevalence, hospitalization, and death from asthma over two decades, 1965–1984. Chest 1987; 91(6):65–74.
4. Kleiger JH, Dirks JF: Medical compliance in chronic asthma patients. J Asthma Res 1979; 16:193–196.
5. Epstein SW, Manning CD, Ashley MJ, *et al:* Survey of the clinical use of pressurized aerosol inhalers. Can Med Assoc J 1979; 120:813–816.
6. Maiman LA, Green LW, Gibson G, *et al:* Education for self-treatment by adult asthmatics. JAMA 1979; 241:1919–1922.
7. Kinsman RA, Dirks JF, Pahlem NW: Noncompliance to prescribed-as-needed (PRN) medication use in asthma: usage patterns and patient characteristics. J Psychosom Res 1980; 24:97–107.
8. Bailey WC, Richards JM, Manzella BA, *et al:* Promoting self-management in adults with asthma: An overview of the UAB program. Health Educ Q 1987; 14:345–355.
9. Bailey WC, Soong S, Brooks CM, *et al:* Demographics co-morbidity, and medication in an asthma population. Chest 1987; 92(Suppl):122.
10. Brooks CM, Richards JM, Bailey WC, *et al:* Subjective symptomatology of asthma in an outpatient population. Psychosom Med 1989; 51:102–108.
11. Haynes R, Taylor D, Sackett D: Compliance in Health Care. Baltimore, MD: Johns Hopkins University Press, 1979.
12. Windsor RA, Green LA, Roseman JR: Health promotion and maintenance for patients with chronic obstructive pulmonary disease. J Chronic Dis 1980; 33:5–12.
13. Clark NM, Feldman CH, Freudenberg N, *et al:* Developing education for children with asthma through study of self-management behavior. Health Educ Q 1980; 7:278–297.
14. Bruhn JG: The application of theory in childhood asthma self-help programs. J Allergy Clin Immunol 1987; 72(No. 5, Part 2):562–577.
15. Thorensen CE, Kirmil-Gray K: Self-management psychology and the treatment of childhood asthma. J Allergy Clin Immunol 1987; 72(No. 5, Part 2):596–606.
16. Cohen J: Statistical Power Analyses for Behavioral Sciences. New York: Academic Press, 1972.
17. Fleiss JL: Statistical Methods for Rates and Proportions. New York: John Wiley & Sons, 1981.
18. Manzella BA, Windsor RA, Bailey WC: Winning against Asthma: An Adult's Self-Help Guide to Asthma Control. Birmingham, AL: UAB Lung Health Center, University of Alabama at Birmingham, 1986.
19. Manzella BA, Brooks CM, Richards JM, *et al:* Assessing the use of metered dose inhalers in adults with asthma. J Asthma 1989; 26:223–230.
20. Morriskey DE, Green LW, Levine DM: Concurrent and predictive validity of a self-reported measure of medication adherence. Med Care 1986; 24:67–74.
21. Richards JM, Bailey WC, Windsor RA, *et al:* Some simple scales for use in asthma research. J Asthma 1988; 25:363–371.
22. Richards JM, Dolce JJ, Windsor RA, *et al:* Patient characteristics relevant to effective self-management: scales for assessing attitudes of adults toward asthma. J Asthma 1989; 26:97–106.
23. Jones E, Sigall H: The bogus pipeline: a new paradigm for measuring affect and attitude. Psychol Bull 1971; 76:349–364.
24. Lowe JB, Windsor RA, Adams B, *et al:* Use of a bogus pipeline method to increase accuracy of self-reported alcohol consumption among pregnant women. J Stud Alcohol 1986; 47(2):173–175.
25. Warner KE, Luce BR: Cost-benefit and Cost-effectiveness Analysis in Health Care: Principles, Practice, and Potential. Ann Arbor, MI: Health Administration Press, 1982.
26. Windsor RA, Warner KE, Cutter GR: A cost-effectiveness analysis of self-help smoking cessation methods for pregnant women. Public Health Rep 1988; 103:83–88.
27. Bailey WC, Richard JM, Soong, S, *et al:* A randomized trial to improve self-management practices of adults with asthma. Arch Intern Med 1990; (In Press).

NOTE

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